ANNALS
OF THE
RHEUMATIC DISEASES

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The Official Journal
of the
EMPIRE RHEUMATISM COUNCIL

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DECEMBER, 1940

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THE EMPIRE RHEUMATISM COUNCIL

Owing to war conditions and the need for economy in paper, two issues only of "Annals of the Rheumatic Diseases" will be published this year, instead of four; one in the Spring and one in the Autumn. Subscribers' payments will be adjusted accordingly. For the same reason, the issue of an Index to Volume One has been postponed.

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CONTENTS

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EDITORIAL

THE Editors regret that publication of the autumn issue of the Annals has been delayed owing to circumstances beyond their control. It is hoped to publish the next number in May, 1941. The Symposium on Fibrositis, which is a feature of this number, has been arranged with a view to concentrating attention on this important aspect of rheumatism and to stimulate further research into ætiology, pathology and treatment. The opinions expressed by the contributors are not necessarily those endorsed by the Empire Rheumatism Council, and may sometimes be at variance with the orthodox conceptions of the disease, but discussion is desirable if progress is to be made. The Editors will be glad to receive and consider further contributions on this subject. Fibrositis is one of the most common causes of invalidity in the Defence Forces, and observations based on work in Service hospitals would be of interest and value.

Not only the Empire Rheumatism Council, but the British community and, indeed, the world at large has sustained grievous loss during the past year by the untimely death of three distinguished leaders in the war against rheumatic disease.

Dr. E. P. Poulton died suddenly in October, 1939, shortly after he had attained to the position of Senior Physician to Guy's Hospital. He was largely responsible for the formation of the Royal College of Physicians' Committee on Chronic Rheumatism which led to the establishment of the Empire Rheumatism Council. It is significant that although his leaning was always to biochemistry, he did much to advance the study of Medical Hydrology and other physical methods in the treatment of rheumatism. He was chairman of the Council of the International Society of Medical Hydrology, and devoted much time and energy to its objects. The Empire Rheumatism Council adds its tribute of

appreciation to a distinguished physician who has passed away while still in the fullness of his intellectual powers.

Dr. Fortescue Fox was an international figure, known and esteemed in all European countries as well as in America. He commenced practice in Strathpeffer after a distinguished student career, but settled in London thirty-five years ago and devoted himself to the study of Medical Hydrology. He was the founder of the International Society of Medical Hydrology and its offspring, the International League against Rheumatism, to which the Empire Rheumatism Council is affiliated. He was an active member of the Royal College of Physicians' Committee on Rheumatism, and a foundation member of the Empire Rheumatism Council. During the last war Fox was active in the treatment of soldiers by physical medicine, in which subject he was a pioneer in placing it on a scientific footing. He published two books on Medical Hydrology founded on sound principles and wide experience. In conjunction with his friend Dr. van Breemen he wrote "The Causation and Treatment of Chronic Rheumatism". His activities were manifold and his circle of friends extended far and wide. His gentle but persistent personality will be greatly missed, and the medical world is the poorer for his death.

Dr. F. G. Thomson of Bath, who died suddenly only a few weeks ago, has left a gap among scientific spa practitioners which it will be difficult to fill. He helped to raise the practice of medicine in the spas from pure empiricism to its rightful place in therapeutics. He wrote, in conjunction with Dr. R. G. Gordon, "The Principles and Physiology of Hydrology," and a book on Chronic Rheumatic Diseases which ranks high in the literature of the subject. He was an original member of the Empire Rheumatism Council and was the first President of the Section of Physical Medicine of the Royal Society of Medicine. He was actively associated with many other movements for the study of rheumatism and its treatment. In addition to his work for the Royal Mineral Water Hospital at Bath, he was Consulting Physician to the British Red Cross Clinic, Peto Place, London.

THE TREATMENT OF SCIATICA, BRACHIALGIA, AND OCCIPITAL HEADACHE

By RALPH STOCKMAN

Sciatica—pain in the distribution of the great sciatic nerve—occurs as a symptom in a number of totally different pathological conditions such as pressure on the nerve elements from a pelvic tumour or bony overgrowth in the vertebræ or sacro-iliac and hip joints, in certain spinal cord lesions, or as a result of the action on the nerve fibres of injury or chemical poisons like alcohol and lead, but such cases, although not uncommon, are seldom seen in practice compared with the large number which are due to chronic fibrositis of the muscles and nerves, and it is to the treatment of these that the present remarks apply.

The essential pathology of fibrositis is now well recognised and agreed upon. Under irritation from one source or another the white fibrous tissue of the muscles, nerves, fasciæ and other fibrous structures undergoes inflammatory hyperplasia in small patches, the affected areas become swollen and œdematous with sero-fibrinous exudation, the fibroblasts proliferate rapidly, numerous minute new bloodvessels appear, and the whole forms a soft, ill-defined, congested little swelling. In the exudate polymorphonuclear leucocytes are entirely absent, with consequent absence of pus formation. At this soft early stage the small inflamed excrescences may resolve readily under treatment or spontaneously, but more frequently they increase in size from continued proliferation of their connective tissue cells, thereby becoming more fibrous and tougher, the perineurium of the nerve twigs ramifying through them undergoes interstitial inflammation and hypertrophy, and the minute arterioles and veins have their walls greatly thickened. Fibrositic thickenings may also begin insidiously, increasing in size very slowly and gradually and attracting no attention for a long time.

On palpation through the skin the larger ones (the size of a small pea to an almond) can be felt as ill-defined swellings along the edges of muscles or in fascia and subcutaneous tissue, but much of the altered tissue is too minute to be detected in this

way and can be located only by its tenderness on pressure. The new formations may be scattered in large numbers all over the body or may be confined to one or a few parts.

The original lesion is most commonly the result of bacterial infection, acute rheumatism and influenza being the most frequent although there are many others, while metabolic products and local injury from cold and overstrain are also capable of starting the whole process. Adhesions are formed in this way, usually by local bacterial infection, and in their origin and structure are exactly the same as other fibrositic growths. Whatever the original cause may have been the end-result is the same: the new formations remain as delicate or more or less dense fibrous tissue well supplied with chronically inflamed nerve twigs and thick-walled bloodvessels. This pathological tissue is extremely sensitive to a large number of disturbing influences such as weather changes, cold winds and draughts, unaccustomed muscular exertion or sudden strain, the toxins of slight febrile illnesses and intestinal indigestion, as well as others known and unknown. Exposed to such irritants the thickened bloodvessels become congested, serum exudes and is not readily reabsorbed, the little areas become swollen and tense, pressure is exerted on the hyperæmic ultra-sensitive nerve twigs and there results pain, or in a lesser degree merely weariness, aching and stiffness. For instance, in a healthy individual unaccustomed exercise (that is, repeated slight traumata from overstrain) irritates the fibrous tissues of the strained muscles to the extent of causing fatigue and aching and stiffness, but, as the nerves are healthy and not hypersensitive pain is not experienced, and, as the bloodvessels are normal congestion and exudation are small in amount and are soon got rid of with or without the help of a hot bath and rubbing. But in a fibrositic subject the effects of unwonted exertion are much more severe and may last for weeks owing to the nerves being hypersensitive and to the pathological bloodvessels failing to deal with the congestion and exudation, and so failing to relieve tension.

By keeping these pathological considerations in mind one is helped to a more precise and individual method of treating cases of sciatica than can be gathered from the textbooks. The advice given in these varies very little from book to book but offers a varied choice, comprising rest in bed, the application of splints, heat, electricity, anodyne liniments, counter-irritants, dry cupping, red rays, ionisation, massage, a great array of palliative and alleged

curative drugs, diet, climate, local injections of cocaine, ether, chloroform, oxygen, water and saline solution, acupuncture and stretching of the nerve. A heterogeneous mass of procedures like this is, however, very little helpful. Many of them are only useful to allay pain, many are useless, and some of value only in selected cases and properly applied. For successful treatment the remedial measures have to be adapted to the pathological conditions present in each patient, and these vary a good deal.

Judged by my own experience in practice sciatic pain most commonly arises from the presence of rheumatic nodules and thickenings in the buttock, and especially in its upper two-thirds. The pain has its origin in the inflamed hypersensitive twigs from the sciatic nerve which traverse the fibrositic areas, and from them it is reflected down the nerve trunk, which is usually healthy and shows no special tenderness on pressure. On palpating over the skin of the buttock (preferably using a lubricant) numerous little swellings can be felt in connection with the fibrous sheaths of the gluteus medius and maximus lying along and just under the crest of the ilium and lower down. They are very sensitive, and can thus be localised by their tenderness to pressure if they cannot be felt. The basis of treatment is massage of the fibrositic areas in the buttock. As adjuvants, heat applied locally for ten minutes previous to the massage renders it more effective and less painful, and anodyne applications locally and analgesics internally have their own proper application. Any other measures are either unnecessary or useless.

The massage is of a simple kind and a lubricant must be freely used. If the patient cannot afford the services of a professional masseur any intelligent person with a little preliminary instruction and occasional supervision can carry out treatment satisfactorily. The early efforts are directed to getting rid of the effusion and congestion in the inflamed areas, and for this gentle but firm effleurage should be given. It is always painful, and the amount of pressure has to be measured by the patient's capacity to bear pain. The swollen congested tissues bleed easily, and at first there may be a considerable amount of bruising and discoloration of the skin, but this does not interfere with treatment and ceases in two or three weeks. The massage should be given daily for twenty to thirty minutes. At the end of three or four weeks the pain it causes has greatly diminished, and from then on it should be given as hard as the patient can reasonably be expected to

bear it. By this time the thickenings have become more defined and can be treated individually with hard pressure, making circular movements with the tips of the fingers and knuckles, but these should always be preceded by a few minutes' effleurage to remove fluid and reduce congestion. The hard pressure either disperses the thickenings or breaks up and destroys their blood-vessels and nerves so that they shrink and harden, cease to respond to irritation, and can no longer become congested and swollen and painful. The massage should be continued until it ceases to cause pain, and this may take two or three months from the start. The prognosis is always good unless the nerve trunk is also involved, when further and different treatment is required.

The other common cause of sciatica is fibrositis of the nerve trunk, the usual site being in the buttock and much less frequently the thigh. It is the nerve sheath which is primarily attacked, but the inflammation spreads into the finer connective tissue of the nerve bundles and fibres, causing interstitial neuritis (neurofibrositis), often with excruciating pain and disability in the limb. As the nerve sheath is a firm, dense membrane closely investing the nerve bundles, when it is acutely inflamed the inflammatory exudate does not readily escape and the tension and pressure on the nerve proper may be severe. An acute attack of this kind leaves in its wake permanent fibrositic tissue which in its turn readily inflames and gives rise to repeated attacks of sciatica. The interstitial neuritis interferes with the axis cylinders, and is thereby responsible for pain and for the numbness, muscular wasting and altered reflexes which sometimes supervene.

The measures to be employed in treating these cases have to be adapted to the local condition. If there is much local reaction in the nerve sheath the indications are to relieve pain, lessen congestion, and promote absorption of the exudate by rest in bed, heat and anodynes locally and analgesics internally. Mild diaphoretics and saline purgation help matters. If experienced and highly skilled massage is available it should be begun at once or as early as possible, and should consist of the gentlest effleurage. Most writers advise that its use should be avoided in the early stages, but it is to be borne in mind that a septic inflammation of connective tissue differs markedly from a fibrositic one. In the former massage would only do harm as the exudation becomes purulent and the tissues tend to soften and break down, whereas in the latter pus never forms and the exudate tends to organise

and form more pathological fibrous tissue. The great object of treatment, therefore, is to get rid of the exudate at as early a stage as possible, and for this massage and gentle pressure by bandaging are the best means available so far. The only contra-indication is that it may be too painful, but that depends largely on the masseur. It soon becomes less painful and should be continued as long as it seems to be doing good. But it is difficult to massage away adhesions and inflammatory tissue buried in the soft substance of the buttock, and recourse may have to be had to surgery to clear the nerve sheath and nerve bundles from their fibrositic tissue. It is in such cases that acupuncture, stretching of the nerve, and injection of fluid or oxygen have been found to relieve pain. They act by stretching adhesions and lessening tension, but their action is uncertain, very often only temporary, and they are not curative, as they do not get rid of the inflamed tissue. Careful surgical dissection is in every respect preferable to such haphazard procedure. Surgery, however, cannot reach the fine interstitial neuritis in the nerve trunk, which must be dealt with by the tedious process of deep massage.

Sciatica coming on after gross injury to the nerve is due to adhesions or a cicatrix, and is best treated by an operation for their removal.

Brachialgia has the same pathology as sciatica and the same principles of treatment apply, but the actual carrying of them out is easier and the result more speedy. On palpation painful fibrositic spots are found in the fibrous structures connected with the trapezius, deltoid, pectoralis major, supra- and infra-spinatus and other muscles, and from the inflamed nerve twigs traversing these spots pain may be reflected to the trunks of the parent nerves—circumflex, median, ulnar and musculo-spiral. The nerve trunks may be quite healthy, and, if so, treatment need be directed only to the painful muscle or muscles. Very often, however, it is one or other of the nerve trunks in the upper arm which is inflamed and tender, usually over a quite small area. The painful areas in muscle and nerve should be identified and massaged. As a rule no other measures are necessary, as, owing to the small bulk of the muscles and the fact that pressure can be made against bone, the massage is very effective.

Headache.—Dull or acute pain in the occipital, frontal or temporal regions is often due to perineuritis of the nerves supplying these parts. The inflamed area is very small and generally

confined to the point where the nerve emerges from deep cover: the supraorbital nerve at the supraorbital notch, the temporal as it crosses the temporal fossa, and the great occipital as it runs superficially in the scalp. Massage at these points can readily disperse the new fibrous tissue. Fly blisters are also useful.

But in the occipital region pain and headache frequently arise from fibrositic areas in the neck and shoulder muscles or in the abundant fibrous tissue of the cervical vertebræ, often, therefore, low down in the neck and at a distance from where the pain is felt. The pain is reflected along the larger nerves from small twigs in the inflamed patches. In order that massage may be accurately applied the whole area must be searched, and if the offending places are superficial treatment is satisfactory, but if they are deep in it is often difficult or even impossible to reach them effectively. As these cases are always tedious patients should be instructed to treat themselves for five or ten minutes daily after having had a course from a professional masseur.

FIBROSITIS: SOME OLD AND NEW POINTS OF VIEW

By C. W. BUCKLEY

Since the word fibrositis was proposed by Sir William Gowers to cover acute lumbago, brachial neuritis and similar conditions, it has come to be very loosely applied and now covers as wide a field as does the word arthritis in the minds of the general public. Stiffness and pain are the distinguishing characters, but vary in degree and their relation to each other: pain may be slight or even absent, and stiffness varies from muscle spasm, or limitation of movement due to fear of arousing pain, to actual contraction of capsular ligaments or adhesions between muscles and other tissues. Critical examination of the current views on the ætiology and pathology seems to be overdue, and an attempt might be made with advantage to discover the relation, if any, between such forms as the lumbago of acute onset and short duration and the chronic degenerative processes seen as a result of overuse in some occupations or even as a mark of advancing years.

It is worth while to recall the description given by Gowers (1904) when he first suggested the name fibrositis. He gave an admirable clinical account in which he says that there is no indication of the formation of inflammatory products, but admits that this is not enough to justify a denial of its inflammatory nature. He pointed out that the pain of lumbago is felt in the afferent nerves, which terminate in the interstitial tissue between the muscle fibres and not on the muscle fibres themselves as do the efferent nerves; the only ending of these afferent nerves is in the bodies called the muscle spindles. These structures appear to belong to the fibrous tissue which sheathes and connects the muscle fibres and not to the muscle itself. They are long structures lying between the muscle fibres; one or more muscle fibres enter each, and two or more nerve fibres, usually one at each end and one in the middle, where they divide and ramify. Normally they give rise to no sensation, and pain, when it occurs, is due to excessive excitation or to an induced excessive sensibility;

it may be caused by compression of the muscle or the adjacent fibres to an undue degree. Fibrositis tends to spread by continuity of tissue, especially to tendinous structures, which themselves contain nerve endings resembling the muscle spindles and consist of an interlacement of nerve fibres enclosed in a capsule, but they are not elongated in shape and contain no muscle fibres; they are sensitive to strain or compression. Gowers described the so-called brachial neuritis as being similar in nature to lumbago but different in seat; the pain arose, he believed, in the muscles and was induced by their contraction or by sudden tension however slight; attempt at movement induces pain so severe that an involuntary contraction of the muscle occurs immediately, fixing the shoulder joint so that the parts appear to be fixed by adhesions.

It is to be noted that Gowers did not attribute the pain of fibrositis to implication of nerves in the inflamed connective tissue but to the sensory function of the muscle spindles and the similar structures in tendons, and it may be presumed in ligaments also. Nor does he mention the idea so frequently suggested that pain may be due to pressure of a nodule on a nerve fibre, which has always appeared to me entirely unsupported by what is known of sensory function. Pressure on the specialised nerve endings whose function is the conveyance of pain sensations may naturally cause pain, but the effect of pressure on a nerve fibre or trunk is not to produce pain of the type characteristic of rheumatism but a " pins and needles " sensation referred to the area of distribution of the nerve, such as is familiar to everyone as the result of pressure on the ulnar nerve at the elbow. Gowers' observations on the function of the muscle spindles appear to be supported by the work of Lewis and Kellgren on referred pain resulting from muscle lesions.

The idea that low-grade inflammation of the connective tissues explains the pathology of fibrositis is hardly adequate, for the cardinal symptoms of inflammation are never marked and often absent. Poynton and Schlesinger define fibrositis as a pathological process underlying various rheumatic disorders both of a chronic and an acute nature. It is a reaction of the cells of the white fibrous tissue which may result from trauma, chill, " allergy," toxæmia, either metabolic or bacterial, or actual bacterial invasion. This definition is wide enough to cover all the possible forms, for under the heading Fibrositis are included conditions as diverse

in ætiology, pathology, and clinical course as are acute rheumatoid arthritis and "malum coxæ senilis."

We owe to Professor Stockman the most complete description of the histological appearances as they are met with in the superficial fibrous tissues and the aponeuroses. He has described inflammatory hyperplasia in larger or smaller patches with numerous fibroblasts, serous or sero-fibrinous exudation, thickening of the walls of the small bloodvessels and nerve sheaths. This description is obviously based on the examination of affected tissues at a later stage and is probably not applicable to the acute stage of lumbago or brachial neuritis with which Gowers was concerned. These changes affect small patches of the fibrous tissues, organisation takes place with the formation of nodules, plaques, and strands of thickened tissue, and adhesions between adjacent structures. These formations are persistent and are apt to be sensitive to barometric changes, dietetic indiscretions, and other influences, and are characteristic of the chronic rheumatic subject.

In view of the scepticism often expressed as to the importance of nodules in the diagnosis and treatment of fibrositis, and the doubts which appear to be entertained, especially in America, as to whether fibrositis is in fact a clinical entity, the point demands further consideration. It has been said that the nodules of fibrositis are perceptible only to the finger of faith, and that they are structures which the physician discovers but the scalpel of the surgeon fails to reveal. It is my own opinion that their importance has been exaggerated. They are common enough, but probably more often symptomless than otherwise. They may be palpated in the superficial fibrous tissues of the buttock, especially just below the iliac crest and in other places in people who have never had any symptoms. Such nodules are painless unless pressed firmly against the underlying bone, which might be expected to cause pain even in normal people; quite frequently small masses of fat are regarded as fibrositic nodules. Attempts to break down nodules of this kind by hard massage are futile and must result in trauma and fresh nodules. Quite different are the localised thickenings in the substance of the muscles, which appear often to be due to binding together of adjacent muscle fibres and spindles by adhesions, the result of earlier inflammatory or allergic attacks, or to local areas of muscle spasm, and these will yield to massage properly applied, which will break down the adhesions, liberate the muscle fibres, and result

in the relief of symptoms. I think it has yet to be demonstrated that a local patch of fibrous tissue, however organised in structure, will give rise to symptoms unless it implicates the muscle spindles or other nerve end organs, or sets up tension between adjacent muscle bundles. These thickenings in the substance of the muscle are usually ill-defined and often difficult to detect, but are generally extremely tender; their importance is less from the point of view of diagnosis than as a guide to the site at which injection of local anæsthetics is likely to have a beneficial effect.

An observation of great importance is that of Gratz (1937), who states that the fascial surfaces of muscles are, like tendons, covered with a layer of mesothelial cells, a functional adaptation of the fascial planes, facilitating movements between the groups of muscles they enclose, thus forming "joints" between the muscles. Fascial adhesions may thus be the cause of pain and limitation of movement, but these symptoms may also result from any interference with the nutrition of these mesothelial cells and their function—in fact, such circumstances as may give rise to synovitis of a joint or tendon sheath, whether dry or accompanied by effusion, which under favourable conditions will clear up without the formation of adhesions. Localised spasm of muscle fibres in the neighbourhood is likely to result, and a nodule or an area of increased resistance may be felt on palpation. This will be associated with pain on attempted movement, but the symptoms will usually clear up completely with suitable treatment or even with rest alone. The crepitus so often met with in the neck apart from any pain or marked stiffness, and which is apt to be attributed to arthritis of the cervical spine, is due to friction between the muscles themselves and adjacent structures and is very like the crepitus in the knees in villous arthritis.

The lymphatic circulation is intimately connected with the fascial spaces, and the fascia derives its nourishment from the lymphatics and not from the blood supply directly. It is important to note that lymph is not simply a transudation from the blood but differs from the plasma in chemical composition and is probably modified by the secretory activity of the tissue cells. It is possible, though difficult to prove, that this excretory activity is selective and eliminates from the blood stream toxic substances of certain kinds, including those of gout, which will exert a deleterious effect on the mesothelial cells. Similarly the result of a local lowering of temperature by exposure to cold, below that

which is optimum for the tissue cells, will if prolonged be productive of pathological effects.

The development of bursæ and even of joints, in an earlier stage of existence, from fissures in the mesenchyme, links together bursitis and synovitis with fibrositis and the lymph in the connective tissue spaces with the synovial content of bursæ and joints, though the latter also contains mucin which appears to be secreted by the cells of the synovial membrane. I submit that a truer conception of fibrositis will be obtained if these points are borne in mind.

The close association of bursitis with other forms of fibrositis is seen in the periarthritis of the shoulder often miscalled brachial neuritis. In this condition we may have an inflammatory condition, sometimes infective in origin, of the fibrous septa of the deltoid muscle spreading to adjacent fibrous structures such as the ligaments of the shoulder joint, the tendons of the supraspinatus, and other muscles, and the subacromial bursa. Or, generally as a result of trauma, acute or chronic, we may find the bursæ primarily affected or the supraspinatus tendon may be frayed or ruptured. The result demonstrates all the characteristic features of fibrositis, especially the muscle spasm in the early stages, the formation of adhesions later, and the referred pain in the distribution of the musculo-spiral nerve so closely analogous with sciatica.

It is interesting to observe to what an extent the conception of a fibrositis of the sheath of the sciatic nerve or the brachial plexus has been abandoned as the referred nature of the pain has been more widely recognised. It would be advantageous to know how often an inflamed nerve sheath has been actually demonstrated by operation or at autopsy in cases other than cellulitis. I suspect that it is uncommon.

That gout may be the cause of fibrositis is denied by some and strongly held by others. Probably this depends on the diagnostic criteria regarded as essential in gout. If, as some hold, the presence of tophi is the only sound evidence of gout it must be admitted that tophi in the common situations are rarely seen unless typical attacks in the joints have occurred, and by no means always then. It would take too much space to argue whether this is a justifiable contention, but there is reason to believe that deposits of biurate of soda occur in other tissues than the cartilages of the ear or the joints or similar sites, and in proof

of this the frequency of gouty deposits in the olecranon bursa may be quoted. If on the other hand a level of uric acid in the blood substantially higher than the normal is admitted as evidence of a gouty state, then gouty fibrositis undoubtedly often occurs.

In a series of cases under my own observation in which estimation of blood uric acid was carried out, in sixty-seven the amount found varied from 4 mg. per cent. up to 7·8; twenty-seven of these were diagnosed clinically as fibrositis; two had had typical gout in addition and showed tophi; thirty were diagnosed as arthritic gout ; and the remainder were other forms of arthritis. The usual normal by the method used is 3·5 in women and 3·7 in men. From these figures, in addition to my own personal observation, I do not hesitate to say that fibrositis is frequently associated with a level of blood uric acid higher than normal, and I regard such cases as demanding treatment, both by diet and drugs, on the same lines as gout of the more orthodox type.

While the forms thus discussed are commonly due to trauma or toxins of bacterial or metabolic origin, the association of these causes with panniculitis is not obvious, and an endocrine factor seems more likely. There are grounds which would justify its separation altogether, for any link between acute lumbago or acute bursitis and the panniculitis of the post-climacteric type is difficult to find. Periarticular fibrositis is, however, often met with in association with panniculitis.

Stockman (1926) has advanced the view that ossifying spondylitis, more usually termed ankylosing spondylitis, originates as a periarticular fibrositis, an important observation which has not received the attention it deserves. He recognises that ossification of inflamed fibrous tissue is rare, though it sometimes occurs in other conditions, and believes it is due to the fibrous tissue cells taking on the function of osteoblasts. I think there are many symptoms in ankylosing spondylitis, especially in the early stages, which present the characters of periarticular fibrositis, and the differences are less than those between spondylitis and rheumatoid arthritis. The question is one which certainly deserves further study.

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THE NATURE OF FIBROSITIS AND THE INFLUENCE OF PSYCHOLOGICAL STATES UPON IT

By R. G. GORDON

Fibrositis is one of the commonest ailments with which the human race is scourged; at least that would seem to be the opinion of British physicians. It is not long since, however, that American physicians denied its existence, though they are nowadays more sympathetic to its recognition. It is interesting to examine the admirable surveys of rheumatic literature compiled by Dr. Hench and his collaborators and notice the growth of recognition and attention paid to fibrositis. The Americans took up the attitude that since there was no cut-and-dried pathology of fibrositis it should not be accorded the dignity of a "disease." In this complaint of lack of defined pathological lesions they have been justified, though Slocumb (1936) has recently described stages in the pathology of this disease—(1) a low-grade inflammatory sero-fibrinous exudate with proliferation of fibroblasts and bloodvessels, (2) local tissue thickenings with the formation of gross subcutaneous nodules, and (3) fibrous contractures or capsular thickenings.

The first person to describe any pathological lesions was Stockman (1920), who succeeded in dissecting out some nodules, and since his work was published British observers have generally regarded the nodule as the hall-mark of fibrositis. Hench (1935) considers that "nodules are the signposts of the disease and may or may not be tender, depending on the stage of the inflammatory activity therein. . . . In my experience biopsy often has been disappointing, and sections of tender muscular spots have often presented little or no histological abnormality and have given negative cultures."

All those who are experienced in the clinical examination of "rheumatic" patients will agree that at least in chronic cases nodules can be discovered if the patient's muscles are sufficiently relaxed and patience is exercised in deep palpation; but all

nodules are not tender, nor do they all have a constant relation to the location of the subjective pains described by the patients. Nevertheless, May (1936) is probably correct when he says that " nodules are always evidence of a previous attack of sometimes subsymptomatic fibrositis. Even the non-tender ones are potentially troublesome." On the other hand, especially in acute fibrositis, if we are honest we must confess that it is not always possible to find palpable nodules in areas in which the patient complains of the most intense pain; the most we can say is that as our finger approaches the tender spot there is a local contraction of muscular fibres, and in such cases biopsy, as Hench says, is often entirely negative.

No doubt the explanation of this in some cases is that the pain complained of is really referred, and the true source of the pain is in one or more of the " spots " which have been described by Kellgren (1938) whence pain is referred to certain areas which he has determined. Nevertheless even Kellgren's " spots " are not always the seat of palpable nodules which can be dissected out.

It would appear, then, that the nodule is the hall-mark of the fibrositic subject, but the tombstone of the fibrositic attack, although without doubt a fresh attack may occur in or about the tombstone.

Dr. Hench has very properly described the efforts of the present author (Gordon, 1936) to discuss the nature of fibrositis as pure speculation supported by no shred of pathological evidence. If, however, there is no demonstrable pathology of acute fibrositis either by biopsy or necropsy, then, if the condition exists which all those of us, even American rheumatologists, who have experienced an acute attack of fibrositis in our own vile bodies will vehemently proclaim, its nature can be arrived at only by critical consideration of the relative probabilities of speculative theories. I propose, then, without shame or apology, to devote the rest of this paper to speculations on the nature of acute fibrositis, in the hope that critical consideration may be given to them by others.

The acute attack of fibrositis is characterised by intense pain apparently induced by (1) sudden strain, (2) wear and tear, (3) chill, or (4) worry, or some combinations of these.

The pain of acute fibrositis is difficult to describe but is, as Sir Thomas Lewis (1938) has said, of the same nature as visceral

pain. So much is this so that acute fibrositis is often diagnosed as thoracic or abdominal visceral disease—angina, pleurisy, colic, or appendicitis; and remedies, whether medical or surgical, are exhibited, the failure of whose action may be the first indication of the true nature of the disability. Lewis considers that muscular pain, being of the deep structure variety, is conveyed to a different area of the sensorium from skin pain. Such pains are often associated, as are for the most part those from the viscera, with quiescence, with slowing of the pulse, a fall of blood pressure, and nausea, the last phenomenon being responsible for the common designation " sickening " which is applied to these, but never to cutaneous pain. This sickening pain is not closely localised, and while, of course, due to stimulation of afferent nerves, the sensation produced is not comparable to that associated with the picking out of pain spots on the skin. Deep pain would seem to be commonly if not exclusively due either to muscular spasm or to expansion within a closed space. That fibrositic pain is often due to muscular spasm is probable, for if the pain is not too acute localised hardenings can be felt which are mistaken for the permanent nodules of chronic fibrositis; but the former frequently disappear and cannot be rediscovered. In addition to this, however, the pain is sometimes wholly, or in some degree, due to dilatation of vessels and exudation of serum into the tissues, for sometimes no " nodule " temporary or permanent can be found, but instead a diffuse swelling; and Slocumb (1936) describes the first stage of fibrositis as a "low-grade inflammatory serofibrinous exudate." It is notable, too, as Burt (1936) has pointed out, that muscles which act in an enclosed space between bony surfaces are specially liable to fibrositis.

A good deal of attention has again been paid in recent months to the efficacy of injections of cocaine derivatives and of quinine in the treatment of fibrositis. This is, of course, nothing new, and the fact that this method of treatment is rediscovered every few years shows that its curative value as opposed to its relieving value is not so great as its enthusiastic advocates would wish their readers to believe. That such injections do relieve acute fibrositis, however, there is no doubt. The chief use of cocaine in medicine is for its action on the superficial pain component which Lewis (1936) has shown is different from the deep component, and its effect on the deep pain may be as much due to its inhibition of general muscular contraction and to its effect in contracting

the arterioles as to any action on specific deep pain nerve endings if such exist. Similarly the local action of quinine (Cushny, 1910) is to inhibit exudation of serum and migration of leucocytes—that is, to diminish swelling in an enclosed space. If such injections, therefore, have anything but a transitory effect it must be because acute fibrositis does not have any permanent pathology, and is a reversible functional condition, which, however, like other functional conditions, may eventually lead to disease processes of more lasting duration which are at least to some extent irreversible, and in the case of fibrositis characterised by the nodule.

It is next necessary to consider the probable action of the four factors which are generally listed as being responsible for fibrositis in producing symptoms.

(i) Sudden strain or acute trauma may result in fibrositis, and in such cases the pathology is reasonably clear. A sudden strain may produce a rupture of muscle fibre with necessarily a small hæmorrhage. Such a rupture is most likely to happen when an unexpected involuntary movement caused by a slip, a stumble or a push causes a sudden stretching of a muscle already contracted. Such an accident causes acute deep pain mostly due to the expansion resulting from the bleeding and increase of pressure in an enclosed space. Later the clot formed may or may not be completely absorbed, in accordance with the vascularity of the tissue into which the hæmorrhage took place. If it took place in fascia or fibrous tissue power of absorption is not great. In such relatively avascular tissue the clot will become fibrosed by the growth of fibroblasts into it and so it will form a nodule. The same process may occur rather less acutely as the result of more gross trauma, which accounts for the fibrositis which tends to develop in the neighbourhood of fractures or dislocations or, for example, in the dorsal tissues of miners who have received deep bruising as a result of a fall of coal on their backs.

The nodule or plaque once formed is apt to be a focus of irritation, and with other "co-operative" causal factors—such as, *e.g.*, chill—a new acute attack of fibrositis may be experienced in its locality, or it may be the seat of the more or less continuous ache of chronic fibrositis.

(ii) The next pathogenic factor is wear and tear, and this is the commonest of all, for fibrositis is most often found in the

middle-aged and in those areas of the body which have been most subjected to strain—the shoulders of miners, the lumbar region of gardeners, and so on. Elsewhere (Gordon, 1936) I have suggested that in such cases the fibrositis is started by the deposition of the products of muscular metabolism which are not removed by the failing circulation of elderly and often hypothyroidic persons. This theory has been criticised because it has not been possible to produce the metabolites out of the nodule by biopsy or necropsy. Yet it is well proved that metabolic waste products are formed as the result of muscular activity and are normally removed by the circulation, and that circulation and elimination in the elderly and hypothyroidic are notoriously slow and inefficient. It has also been said that there is no justification for regarding these elderly sufferers from fibrositis as being hypothyroidic without a long series of controlled observations on their basal metabolism; but it is suggested that even without submitting the patient to the undoubted discomfort of metabolic estimation the clinician may recognise the overweight, lethargic, fibrositic with falling hair, dry cold skin which never sweats as being deficient in thyroid secretion. In such cases, then, we have a non-microbic focus of irritation with reactive congestion round it, with dilatation of vessels, exudation of serum going through the three stages described by Slocumb till the fully formed nodule is discoverable which calls for treatment by heat and massage. In the early congestive stage the condition may be relieved by injection of cocaine or quinine, or again when the deposit of new irritative material in the neighbourhood of old nodules results in an acute exacerbation similar relief may be experienced, but in such patients it is not my experience that anything like a permanent cure results from such methods or indeed from any method, though heat and massage will give longer remissions than any other form of treatment. The influence of the microbic theory of disease and the cult of focal infection is still so strong that in all probability it would be regarded by many as the rankest heresy to suggest that infection, and especially focal infection, is not a potent cause of fibrositis, and it would be absurd to suggest that it never is so. It is as difficult to prove as to disprove any theory of the cause of fibrositis, but to the disappointment of the focal infection fanatics it is seldom if ever possible to recover micro-organisms from fibrositic lesions, nor does the removal of septic foci always,

or I would maintain even often, make any appreciable difference to fibrositis. Immediate relief is not unusual, but anything like a permanent cure from removal of such foci is definitely unusual. This immediate relief is not necessarily due to direct effect on the nodule or local condition, but may be due to effect on general circulation or even on the sensorium in the same way that protein shock and allied methods, such as some forms of vaccine therapy, may act. The real difficulty, of course, is that the majority of elderly people suffer from some degree of fibrositis, and it is seldom difficult to find some sort of focal infection in such people; but the juxtaposition of these conditions does nothing to prove cause and effect.

(iii) The next supposed cause of fibrositis is chill, but no one has explained why sitting in a draught should be followed by an acute stiff neck or a sudden onset of lumbago; but however much the scientist may protest that it can't happen because there is no demonstrable pathology, the answer of the unfortunate subject would probably resemble the comment of Dr. Johnson on Bishop Berkeley. Any explanation must be highly speculative, and therefore no apology is made for the following phantasy. It is assumed that chill and chill only is productive of the disability, and this does seem to be the clinical story in the case of certain susceptible people. It is interesting to note that there is a definite analogy in another common malady—namely, chilblains. Chilblains occur in certain susceptible persons with poor distal circulation on exposure to cold, and manifest themselves as relatively small circumscribed areas of skin and subcutaneous tissue, which are the seat of vascular dilatation and exudation of serum, which is accompanied by the normal type of unpleasant cutaneous sensation—namely, itching and sharp pain.

The only possible explanation of the chilblain is that in certain people who may be susceptible by reason of vitamin deficiency, endocrine anomaly, or other unknown cause, there are "loci minoris resistantiæ" in the peripheral part of their vascular network which under the influence of cold are the seat of dilatation and serum exudation.

When considering the effect of chill in relation to fibrositis we may notice certain facts. It is not everyone who sits in a draught who gets an acute stiff neck or an acute lumbago, therefore we are dealing with susceptible persons. The clinical symptoms and signs consist of the deep form of unpleasant sensation—viz.,

indescribable " sickening " pain (*vide* Lewis, 1938) which tends to radiate and be referred in certain directions (*vide* Kellgren, 1938) and local areas of tenderness and perhaps slight palpable swelling. If any pathology exists at all it consists of dilatation of vessels and exudation of serum (Slocumb, 1936).

Have we then in such cases a condition occurring in deep fascial layers and muscles analogous to the chilblain, the symptoms being translated, as might be expected, into terms of deep sensation as opposed to superficial sensation ?

The whole question of " the susceptible person " and the " unstable vascular network " brings us to the discussion of the next commonly accepted cause of fibrositis—namely, " worry ", since prolonged emotion is liable to disturb the autonomic control; but before discussing this it might be remarked that an interesting and fruitful line of enquiry is opened for those who have access to large numbers of patients. They might undertake to investigate whether there is any analogy between biochemical findings in those susceptible to chilblains and those susceptible to such fibrositic manifestations as acute stiff neck. The difficulty of course is that neither chilblains nor acute stiff neck is a sufficiently serious disability to warrant retention of many such patients in hospitals for periods long enough to carry out such investigations or to encourage such patients voluntarily to submit to the necessary laboratory discipline.

(iv) Fibrositis due to psychogenic causes will of course include those said to be due to " worry " in the ordinary category of ætiological factors.

Doubtless, as Halliday (1937) has pointed out, there are a number of patients who complain of fibrositic-like pains which are purely symbolic in nature as expressive of a state of mind, and are being consciously or unconsciously used by the patient as a protest against the situation in which he finds himself or as an escape from the obligations which are imposed upon him by circumstances. Since some degree of fibrositis is almost always discoverable in the form of nodules which may be " subsymptomatic ", it is a matter for argument whether the pain is entirely symbolic—*i.e.*, originating exclusively in the sensorium—or whether the patient is using a mild degree of discomfort either consciously or unconsciously to achieve an object; but it should be stressed that the vast majority of such patients are not deliberate malingerers and are therefore acting unconsciously.

For example, a chief petty officer complained of pain in the back. He was a perfect example of Halliday's type of case, for he had finished his twenty-five years' service and so was eligible for full pension. He had a sedentary job waiting for him in civil life which did not involve the strenuous physical exercise which was expected of him in the Navy and which he declared he could not do on account of his pain. He felt he had served the Navy well in all parts of the world, but did not enjoy the war or appreciate the constant attention of enemy bombers. On examination he certainly had some fibrositic nodules such as might be expected in any man of his age who had lived an active life, but they were very useful to him in achieving discharge from the Service, and the treatment of this man would obviously have to be directed to inspiring him with greater martial ardour rather than bothering very much about the nodules in his back.

The next group are those who complain of fibrositic pain not primarily because they are definitely using it for the purpose of avoiding a situation, but because their whole sensorium is more sensitive to incoming stimuli than normally is the case, and therefore the pain from a relatively mild fibrositis is more intensely felt. Such a case was a young soldier who had been subject to the most intense aerial bombardment and as the result of this was suffering from a severe degree of acute anxiety neurosis of the war service type. He stated that for some time he had been accustomed to suffer at times from "rheumatic" pains in his back, but had never given much heed to them and had never had to leave work on account of them. Since the bombardment, however, the pains in his back had become very intense, so much so that he could not be comfortable lying in bed because of them, and they contributed to his insomnia, which was, as usually is the case, a prominent symptom of his anxiety. Examination disclosed a few tender nodules in his lumbar and gluteal muscles. His anxiety was accompanied by a markedly increased sensitivity to sound, light, and touch, and treatment directed to his anxiety by abreaction and reassurance diminished this sensitivity with a corresponding diminution of his backache. If, however, the anxiety of such a patient is not completely removed he is apt to cling to his most obvious and as he perhaps hopes his most objective disability, the "rheumatism of his back," since in his mind this alone protects him from a future situation which must still be fraught with anxiety. This is the

situation which so often prolongs disability in peace-time workmen's compensation cases, since they are faced with the two questions: Is there work for me if I am declared fit ? and if there is work, shall I be able to continue to carry it on or shall I in three months find myself given the sack with no prospect of re-employment? This makes it so essential that proper rehabilitation and guarantee of employment should be provided for such cases if they are to be got back to work.

The next group is much more difficult to define and much more difficult to treat successfully. I have dealt elsewhere (Gordon, 1940) with the influence of anxiety states on vascular and other vegetative function which will produce sensitivity to atmospheric conditions and vascular instability. Reference has been made to susceptible people who are especially prone to chill, and anyone who has large experience of persons suffering from so-called sciatica, which is most commonly due to a gluteal fibrositis, will realise how often the trouble is said to be due to cold, sitting on wet grass, etc., and how often the sciatic patient belongs to the class which includes all grades from those who are described as highly strung to those suffering from a frank anxiety neurosis. Sciatica is a very painful complaint, as all sufferers from it will agree, and is not as a rule quickly cured; but many people take much longer to get better than might be expected by the optimistic physician, and the reason is that a vicious circle is set up by the constant pain and lack of sleep which sensitises the sensorium so that the intensity of perception of pain is increased and with it the anxiety which may still further increase the fibrositic reaction by its influence on the vegetative controlling centres in or about the hypothalamus. Nor is this anxiety lessened by the enthusiasm with which consulting physicians and surgeons talk about sciatic neuritis and forcibly stretching the nerve, of sacro-iliac strain and elaborate manipulation, and subluxation of the nucleus pulposus and operations to remove these mysterious pressures, which any neurologist would expect to paralyse the nerve roots and thus abolish the pain. Probably after all the best treatment for these sciatic patients is masterly and sympathetic inactivity and skilful neglect of the physical side of the trouble combined with adequate attention to the mental side.

It is not, however, only in the gluteal region that the frigisensitive neurotic person is prone to fibrositis, and the more

youthful type of fibrositic patient is often of this type as opposed to the more phlegmatic hypothyroidic elderly type whose fibrositis is associated with much more definite nodule formation. The pathology of this, as has been suggested, is the sequence of poor circulation and elimination with failure to deal with irritative metabolites. In the younger patient, on the other hand, it is much more difficult to discover well established nodules, and the attacks may be and generally are much more acute. It is suggested that in such patients circulation is irregular and uncertain and local spasms of arterioles or dilatation of capillaries are apt to occur, especially under the influence of cold or chill. On the other hand, in some cases, as Alkan (1930) has suggested in the case of smooth muscle, local muscular spasms may lead to anæmia, stasis, ischæmia and subsequent nodule formation. Either of such processes would result in muscular pain, and, as Lewis (1938) says, this is not a stimulating but a depressing pain, and depression, if it is not a true psychotic depression, increases perception of pain. To such people pain is always a source of alarm and anxiety and links up with unconscious fears and self-accusations, associated with feelings of guilt calling for punishment, and so a vicious circle is established in which the incoming pain sensations intensify the already existing or potential anxiety state, while the anxiety state may well by its disturbance of endocrine and hypothalamic activity still further increase the sensitivity and vascular instability which is the matrix of the fibrositis. Cases in which even conscious anxiety is expressed by an attack of acute fibrositic pain are by no means uncommon, and the muscular pains which are met with in those who have to nurse a beloved relative through a serious illness are not all due to the standing and bending involved. The pain in such cases may become hysterical and be used to avoid the painful situation, but this is by no means always the case, for many such patients emphatically do not avoid the situation.

It may be observed, then, that psychogenic factors may be responsible for a fibrositic type of pain and may intensify or even produce an acute fibrositis which may develop into a chronic and partially irreversible condition with typical nodule formation. It is suggested that it is extremely unlikely that any one specific cause will be found to be responsible for a " disease " fibrositis, and that what we call for convenience fibrositis is by no means always inflammatory as is suggested by the suffix " itis," but is

a tissue reaction to a number of pathogenic causes. The nature of the particular pathogenic agent should be considered in each individual case, since only so can appropriate treatment be indicated, and amongst these agents disturbances of the *psyche*, acting either independently or in conjunction with disturbances of the *soma*, must not be neglected.

Summary

Although fibrositis is so common it is still poorly understood. This is because it is so difficult to define an established pathology, chiefly since, at least in the acute form, there is no demonstrable pathology and the nodule is often the tombstone of a previous attack rather than the hall-mark of a present attack.

An exposition of the nature of fibrositis must therefore be largely speculative, and it is suggested that four commonly listed causes of fibrositis—sudden strain, wear and tear, chill, and worry—may represent four different pathological processes. The sequences may be:

Sudden strain—hæmorrhage—organisation of clot—nodule formation.

Wear and tear—deposition of metabolites—failure of removal and elimination—reactionary formation of nodules.

Chill—local vascular disturbances analogous to those of chilblain with or without permanent nodule formation.

Worry—psychogenic muscular pain or fibrositis of three sorts: (1) when pain is used as a symbol of emotional discontent, (2) when the whole sensorium is oversensitised as a result of acute emotional disturbance, (3) when chronic emotional disturbances produce endocrine and autonomic disturbances through vegetative controlling centres in the hypothalamic region which are followed by fibrositis.

The relation to these speculations of the nature of muscular pain and the effect of local analgesic injections is discussed, as is the failure of the microbic and focal infection theories to establish themselves as the universal cause of fibrositis.

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FIBROSITIS AND PAIN

By J. B. HARMAN

The object of this paper is to draw attention to a type of pain which is not yet generally recognised as sometimes arising from fibrositis, and which is usually attributed to disease of viscera if it occurs in the trunk, or to brachialgia or sciatica from other causes if it occurs in the shoulder or buttock. The present time is opportune, partly on account of the revival of interest in treatment by anæsthesia, which provides a convenient method of checking the diagnosis, and partly because recent work has demonstrated the circumstances in which this type of pain may be felt.

The symptoms usually associated with fibrositis vary from a vague stiffness to an agonising burning or stabbing pain, and in all cases the pain is made worse by movement of the affected part and by cold and damp, etc. It has a "sharp" quality such as is found with painful lesions of the superficial structures of the body, however produced, and, like them, is easily localised. On the other hand, the type which we wish particularly to consider has a "dull" quality, ranging from a mere ache to an intolerable bursting or cramp, such as may arise from the viscera; and, more important still, the pain is so poorly localised that its greatest intensity may be felt at some distance from its point of origin.

Cases have recently been described[1] in which a dull pain was felt in the front of the abdomen or chest, which was nevertheless due to fibrositis of the back. These cases were all referred with a diagnosis of some visceral disease on account of the site of the pain and the tenderness, muscular rigidity and skin hyperæsthesia which were present. In no case was there any demonstrable disease of the organs under suspicion, and a detailed history showed that, although the site and quality of the pain were consistent with the original diagnosis, yet its exacerbation and relief were dependent on position and movement of the trunk, indicating a somatic origin; while its course and frequent association with other rheumatic conditions suggested that fibrositis was the

cause. This diagnosis proved to be correct, because it was possible to relieve the symptoms by injecting procaine into tender spots which could be discovered deep in the musculature of the back.

Pains referred from the back are well recognised mimics of abdominal disease, and, in the absence of any definite lesion such as tuberculosis or malignant deposits in the spine, are usually attributed to osteo-arthritis of the spine, or to neuritis in the sense of an inflammation of the nerve trunk. The cure by injections of procaine showed that the pain was not due to any of these causes, for the needle was not placed anywhere near the nerve trunks or the joints, nor indeed was osteo-arthritis present in the majority.

Since it can be demonstrated that fibrositis may cause either a sharp well-localised pain, or a dull poorly-localised one, it is pertinent to enquire what determines the difference. Kellgren[2] has studied the pain sensation resulting from the injection of small amounts of hypertonic saline into various somatic structures. He found that injections into the skin, fascia and subcutaneous periosteum gave the well-localised pricking or burning sensation with which everyone is familiar from superficial injuries. If, however, the injection was made into the deep muscles or ligaments, the pain had a dull quality such as is felt from a sprained ankle or dyspepsia, or in angina pectoris, and was not accurately localised to its source, but felt diffusely throughout the corresponding spinal segment. The maximum pain sensation was in many cases some distance from the lesion, tending towards the periphery of the segment, so that the pain might be felt in the front of the body or distally in the limbs even when the injection was in the interspinous ligaments. The pain was accompanied by hyperæsthesia of the skin and muscles and by spasm in the area of its distribution. So far as is known, these two types of pain perception, the superficial and the deep, do not depend on the nature of the lesion, but are determined entirely by the depth of the structure that is injured. It may be, therefore, that a study of the quality of any pain can only indicate the approximate depth of the lesion from the surface, while the diagnosis of the nature of the lesion must depend on the periodicity of the pain, its clinical course and associated symptoms. These modes of pain perception are not, however, two separate categories, but two extremes of a continuous series, for a lesion may be situated in a mid-posi-

tion, or extend in depth from the surface, giving rise to mixed symptoms.

The sort of symptoms which would result from fibrositis of any part of the back may now be discussed. The more superficial forms with local pain anywhere between the sacrum and the occiput need not detain us. The symptoms of deep fibrositis of the trunk with thoracic or abdominal pain have already been referred to, and the headache from nuchal fibrositis and the radiation of lumbago into the side of the buttock or front of the thighs are too well recognised to need further discussion.

The difficulties arise in considering deep lesions around the shoulder or buttock, for these may give rise to syndromes of brachialgia or sciatica. Fibrositis in these situations would on *a priori* grounds give a pain of segmental distribution in the limb, varying in intensity from a poorly-localised ache to an agonising soreness. There would be tenderness of the muscles in the area of reference and hyperæsthesia of the skin, which by analogy with the abdominal cases mentioned would pass over to analgesia with time. There would be a position of the limb in which pain would be minimal and deviation from this would increase it by stretching hyperæsthetic structures. Direct irritation of the fibrositis in the shoulder or buttock by pressure or movement would result in exacerbation of the pain both locally and throughout the affected area. It would be found that some persons were prone to these and other recognised rheumatic conditions, the individual attacks being precipitated by damp, infections and muscular strain.

So far this reconstruction would apply equally well to brachialgia or sciatica, whatever might be their supposed cause, except that the hypotonia, wasting and weakness of the muscles and diminution of the reflexes in the chronic cases have not been accounted for. Experimental pains can give no information here because they last only a matter of minutes, and are incapable of inducing the signs of chronic disease; nor can a study of referred pains in the trunk provide an analogy, because changes in muscles and reflexes would not be manifest in the abdomen or thorax owing to the anatomy of the part.

In considering further the claim of fibrositis to produce brachialgia and sciatica, it will be helpful to compare it with the evidence on which other suggested causes are based, and to examine the validity of their special diagnostic criteria. It is

a commonplace of medicine that even such distinct conditions as spinal tumours and malignant or other deposits in the pelvis may in their early stages give symptoms identical with "idiopathic" sciatica. This being the case, it is unlikely that the vaguer entities such as inflammation of the nerve trunks or their roots, strain of the sacro-iliac joint, fibrositis of the shoulder and buttock and so on, will be distinguished clinically with ease. The notable sensory changes, wasting of the muscles and diminution of the tendon reflexes, have been held to indicate that in some way the nerves themselves must be affected. But it has already been shown that this does not hold good for the sensory changes, and muscles may waste, and to a lesser extent reflexes disappear, in chronic inflammations such as rheumatoid arthritis where there is no question of a primary neuropathy; while it has long been known that the muscle wasting of sciatica does not follow the distribution of the peripheral nerves, but that of the segmental supply. Further, if these signs do indeed indicate direct nerve involvement, then strained sacro-iliac joints, as well as fibrositis, must be excluded from the list of possible causes of sciatica.

Various specific signs have from time to time been described as being diagnostic of a particular lesion. Thus the pain resulting from straight leg raising has been held to show that the nerve is diseased because its inflamed fibres may be stretched in this way; but other structures are disturbed besides the nerve, and the pain might equally well be due to stretching of hyperæsthetic muscles and fasciæ, or fibrositis anywhere in the buttock, or to direct movement of a diseased joint. This argument applies to similar diagnostic procedures, for it is difficult to move any part of the body without causing pain, as a patient with severe sciatica will testify. Tenderness of the nerve trunk itself is often described, with the implication that it is the seat of the primary inflammation, but here again it is difficult to be certain that only the nerve is being stimulated when other structures are sensitive, and equally tender places far from the nerve can often be demonstrated, especially if they are sought for with the same confidence and care. Moreover, even if it be admitted that the trunk is tender, it has still to be shown that this means an inflammation there, and not merely a hyperæsthesia predominantly of the nerve trunk rather than of the muscles or fasciæ.

This leads to the conclusion that the distinction by clinical

examination between the various putative causes of sciatica or brachialgia is very difficult, for those procedures which have been held to be diagnostic of one apply equally well to another, and any difference of opinion is largely a difference of interpretation. The causal lesion of any given case can therefore only be decided by more direct examination. This can be done unequivocally, though by no means simply, for ruptured intervertebral discs, and it is a matter for future experience to show how frequently these occur. The fact that the sciatic trunk is sometimes inflamed has been demonstrated by operation, and it may be assumed that procedures directed to remedying this, such as epidural injection or distension of the sheath with saline or oxygen, do, when successful, confirm the diagnosis. Per contra, their notable failures suggest the opposite. The pathology of strained sacro-iliac joint is purely hypothetical, depending on an analysis of various movements which bring on the pain, and while indeed it may occur and cause sciatica, there is no direct means of examination to prove this to be the origin of a pain which yields to treatment by manipulation or support.

Neither is the claim for fibrositis supported by any direct examination, nor from the nature of the disease is it likely to be. It rests mainly on the experience that brachialgia and sciatica can sometimes be cured by injecting tender spots with procaine, which is a known means of curing fibrositis, and it is difficult to explain on any other hypothesis. The results are clearer with brachialgia, for the shoulder girdle is a simpler structure, and though they are not so convincing in the case of sciatica, an estimate of the frequency of fibrositis based on this test will probably be too low, because procaine injections are by no means a panacea even for obvious superficial fibrositis, and it is difficult to be certain that the correct spot has been injected in so large a structure as the buttock.

Nevertheless it would be rash, on such slender evidence alone, to claim that fibrositis is a general cause of these syndromes, especially in a condition where so many different conclusions have been deduced from the same facts, and it is for this reason that the attempt has been made to show that the suggestion is based on other grounds than the mere results of injection, and to indicate its relationship to other causes and to the perception of deep pain in general. From the point of view of pathology the distinction between the various causes of brachialgia and

sciatica (ruptured intervertebral disc excluded) is more apparent than real, for whatever structure may be responsible it is agreed that it is the fibrous tissue that is affected, whether this fibrous tissue happens to be in a nerve sheath, a joint capsule, a ligament or a fascial plane.

There is, however, considerable practical importance in knowing where the affected tissue lies, if any more specific therapeutic measures are to be employed than rest, heat, and analgesics, for the site will decide the choice between procaine injection, epidural injection, distension of the nerve sheath, mobilisation, or support. Since it frequently happens that the responsible lesions cannot be diagnosed clinically with any accuracy, it would be very helpful if some estimate could be made of their relative frequency, for the appropriate methods could then be used according to the probability of any given lesion being present, rather than haphazard, or according to the fashion of the moment.

The conclusion to be emphasised is that although the main symptom of fibrositis is pain, either of the superficial or deep type, with its associated changes in muscle tone and reflexes, yet no analysis of its *quality* will establish a diagnosis, for these phenomena are not diagnostic of particular lesions, but of modes of pain perception which are dependent on the site of the lesion. This conception of the unity of pain response will correct the tendency to attribute signs and symptoms to one particular lesion which are in reality common to many, and also, by emphasising the many possible causes of pain, will prevent attention from being focussed too exclusively on the most obvious organ of any given segment, whether it be the heart, the appendix or the sciatic nerve. The diagnosis of fibrositis, as of any other lesion, can only be fished out of this common pool by an analysis of the *variations* of the pain—of its periodicity, of what makes it better or worse, and all those factors which together constitute the natural history of the disease, coupled in favourable cases by the discovery and cure of the local focus of irritation.

Summary

The type of pain arising from fibrositis of deep structures is not yet generally recognised. The symptoms of deep fibrositis of the trunk are described and compared with similar experimental pains. The claim of fibrositis to produce the syndromes of

brachialgia and sciatica is discussed and compared with the evidence in support of other causes. The claim is supported by the results of procaine injection in some cases. The syndrome of pain from deep fibrositis is similar to that due to any lesion in a like situation. The diagnosis of fibrositis must therefore rest on the natural history of the disease and the response to treatment.

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DIFFERENTIAL DIAGNOSIS OF PERIARTICULAR FIBROSITIS AND ARTHRITIS*

By C. H. SLOCUMB

Periarticular (capsular) fibrositis is much too commonly mistakenly diagnosed as arthritis. At first there may be little or no pain when joints are at rest or in moderate use. Pain or stiffness may be noted only during the first few moments on awakening or after the joints have not been used for a while, or when the joints are used excessively, or when the capsule is stretched. The patient may have to hunt for soreness by twisting his joint sidewise, squeezing it, or flexing it forcefully to convince himself of the actuality of, and of the situation of, pain. After a short experience with these rather intangible symptoms, the patient may note their gradual disappearance.

If the disease progresses pain becomes more constant, chronic aching may appear, and tenderness becomes less elusive. Joints feel stiffer; yet relatively full, though painful, motion may still be readily accomplished. Generally definite swelling or hydrops does not appear. However, capsular thickening may appear. Usually there is little, if any, redness or significant muscular atrophy, and the roentgenograms represent a normal condition. It may be difficult for the patient to limber up on walking. Setting-up exercises, a hot bath, or acetyl-salicylic acid may be required to produce sufficient limberness. Damp or cold weather may make the stiffness or aching worse. Marked fatigue and nervous exhaustion may appear. In spite of these symptoms, physical examination may not disclose anything essentially abnormal. Sometimes gross fibrous nodules are found over the elbows, knuckles, or about the gluteal, sacro-iliac, or occipital regions.

These symptoms may be present for a few days or weeks, for months or years. Vagrancy and variability in the intensity of

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symptoms are characteristic, much more so than in atrophic arthritis.

Generally the affection disappears eventually, leaving no deformity of joints. When contractures and deformities do occur, they result from periarticular and not from intra-articular disease.

In the absence of direct pathologic proof, abundant indirect evidence demonstrates the persistent integrity of the joint cavity in cases of fibrositis. In periarticular fibrositis the disease is at the joint, not within the joint. In infectious arthritis, however, there is fibrositis plus synovitis, chondritis, and osteitis.

Clinical Differential Diagnosis ; Comparative Symptomatology.—Periarticular fibrositis and atrophic arthritis have several features in common: incidence by age, manner of onset (acute or chronic), relative incidence by sex, bodily types affected, and prodromal symptoms (fatigue, chilliness, nervous exhaustion). However, certain other features are sufficiently characteristic of each to be helpful in diagnosis.

Joints.—The clinical course of periarticular fibrositis differs from that of atrophic arthritis. In atrophic arthritis there is a tendency to polyarticular invasion. In periarticular fibrositis, although many joints are sometimes affected, often only one or two large joints or several small joints are affected. Articular tenderness is to be expected in cases of atrophic arthritis; in periarticular fibrositis generally little or no tenderness is present.

In atrophic arthritis stiffness of joints is generally objective as well as subjective; in periarticular fibrositis it is frequently only subjective. The symptoms of " jelling ", stiffness of a joint during rest, may be the first or only symptom. Atrophy of muscles is commonly seen in cases of atrophic arthritis but is generally absent in periarticular fibrositis.

In atrophic arthritis three factors produce the appearance of articular swelling: (1) hydrops from the synovial reaction, (2) fibrous thickenings of periarticular tissues, and (3) atrophy of muscle. In periarticular fibrositis there may be slight thickening of periarticular fibrous tissue, but factors 1 and 3 are absent; hence swelling is not notable.

Motion of affected joints usually causes pain to patients who have atrophic arthritis. In periarticular fibrositis there may be no pain on ordinary motion, but pain may be present with unusual, forced, or lateral motion which stretches the capsule. Arthritic

joints often feel worse after moderate exercise. Joints affected with fibrositis are frequently subjectively better after moderate exercise. They may hurt only the first few minutes of the day and feel all right thereafter. In atrophic arthritis there are few complete remissions or even complete intermission in symptoms; although symptoms vary in intensity there is generally a constant minimum of discomfort. In periarticular fibrositis, complete remissions, particularly short complete intermissions, are frequent. Patients who have periarticular fibrositis experience much more marked variability in intensity and persistence of symptoms than do those who have atrophic arthritis. However, fibrositis may produce symptoms that remain chronic for a long time. In atrophic arthritis there is little or no tendency to migration of symptoms. In periarticular fibrositis different joints are often involved in successive bouts.

Systemic Manifestations.—Atrophic arthritis is a disease of joints attended by many systemic manifestations. Common findings are slight fever, subnormal cutaneous temperature, tachycardia without fever, lowered blood pressure, anorexia, and significant loss of weight. In periarticular fibrositis these are usually absent.

Fatigue and nervous exhaustion are frequent attributes of both diseases, but in atrophic arthritis they are often submerged since the patient does not exert himself much. Patients who have periarticular fibrositis are usually quite active in spite of their pains; hence fatigue and nervous exhaustion are generally marked and are often notable for being out of proportion to the objective evidence of disease.

Weight.—Sixty-two per cent. of patients who had arthritis lost weight; 47 per cent. of them lost more than 10 pounds. Only 7 per cent. of those who had fibrositis lost more than 10 pounds.

Roentgenographic Alterations.—Of one hundred patients who had fibrositis, seventy-eight, who had suffered a total of 284 years of their disease, presented no intra-articular roentgenographic alterations, although thirty-seven patients were forty-five years of age or older. In 22 per cent. of cases there were hypertrophic changes, generally slight, interpreted as evidence of coincident, unrelated, senescent arthritis, since most of the patients were between fifty and sixty-six years of age. Roentgenograms of some patients were negative after ten to twenty-five years of periarticular fibrositis.

In contrast, in 86 per cent. of cases of atrophic arthritis there was definite roentgenographic alteration. Although half of the hundred patients had had their disease less than a year, only 14 per cent. of the entire group gave negative roentgenograms.

In a given case any intra-articular roentgenographic alteration speaks at once for arthritis (with or without fibrositis). If roentgenograms are negative for six to twelve months, without other definite signs of arthritis, one may favour a diagnosis of periarticular disease. In a case of short duration in which roentgenograms are negative, other data must be relied on for differential diagnosis.

Laboratory Differential Diagnosis.—Regard for clinical differences permits accurate differential diagnosis without recourse to the laboratory, when symptoms have occurred for months or years without objective evidence of intra-articular disease. In the early weeks of a given case, however, differential diagnosis is not easy. Laboratory data may then be helpful. One hundred cases each of fibrositis and atrophic arthritis were studied; cases were selected only to ensure that enough early and late cases of each would be analysed. In the cases of fibrositis the disease had lasted from four weeks to forty years; in arthritis from three weeks to eighteen years.

Sedimentation rate of erythrocytes:—The greatest laboratory differences were observed in estimations of the sedimentation rates of erythrocytes. In cases of fibrositis the average sedimentation rate (at the end of an hour) was 11·6 mm. The rate was normal in 73 per cent., but slightly altered (16 to 25 mm.) in 16 per cent., and significantly altered (25 to 32·5 mm.) in only 11 per cent. The rate was never more than 32·5 mm. Of patients who had rates of more than 20 mm. all but one had had fibrositis more than one year, most of them more than five years.

In contrast the average rate in cases of atrophic arthritis was 71·5 mm. Although 25 per cent. of the arthritic patients had had their disease less than six months, only 3 per cent. had normal rates. Only 6 per cent. had rates between 16 and 25 mm.; 91 per cent. had rates of more than 25 mm. In 67 per cent. of cases the rate was more than 50 mm., and in 23 per cent. it was between 100 and 150 mm.

In atrophic arthritis altered rates were generally noted early, even within the first three to six weeks of the disease. Among

six patients who had fibrositis of six months' duration or less, only one had an elevated rate (23·5 mm.).

Blood counts:—Little or no help in differential diagnosis is obtained from total leukocyte counts or erythrocyte counts. Estimation of hæmoglobin gives clearer evidence of anæmia. In the blood of only 5 per cent. of patients who had fibrositis was the concentration of hæmoglobin less than 13 gm., and in that of only one (a patient who had menorrhagia) was the concentration less than 12 gm. In 42 per cent. of patients who had arthritis, the concentration of hæmoglobin was less than 13 gm. and in 19 per cent. less than 12 gm.

Combination of Abnormal Findings. — Elevation of the sedimentation rate and reduction in the erythrocyte count, in concentration of hæmoglobin, or in weight are, of course, not specific for these two diseases. Hence a patient who has periarticular fibrositis might exhibit one of these alterations because of some other cause than his rheumatism. It is well therefore to note that of the hundred cases of fibrositis, in ninety there were either none of these alterations or only one alteration. Those exhibited by the remaining ten patients were practically never sufficiently impressive to be definitely misleading. In contrast the majority of the patients who had atrophic arthritis exhibited two or more alterations that were not slightly but definitely abnormal.

In a given case, therefore, if there is only one slight abnormality in the laboratory findings, too much significance must not be attached thereto. It may be the basis of a guarded, tentative diagnosis of arthritis rather than of a final one of fibrositis. When in doubt, however, I favour making the tentative diagnosis of fibrositis rather than making a more pessimistic appraisal of the condition and running the risk of making a diagnosis of " arthritis " in a case in which it probably does not exist.

Summary and Conclusions

In any given case, then, the differential diagnosis between periarticular fibrositis and atrophic arthritis is based on a survey of certain direct and certain indirect evidence for or against the presence of intra-articular disease. Direct evidence in favour of intra-articular disease is afforded by the appearance of synovial hydrops and of roentgenographic evidence of disease. Less direct evidence is afforded by the appearance of the joints;

notable redness, tenderness, muscular atrophy, and definite swelling speak in favour of arthritis, as opposed to disease that is exclusively extra-articular. Indirect evidence is afforded by a history characteristic of one or the other type and by certain laboratory data, particularly the sedimentation rates of erythrocytes and the estimation of hæmoglobin. The weight curve also is significant. Alterations in these data, particularly in two or more factors, if the magnitude of the alterations is beyond a certain maximum generally seen in cases of fibrositis, speak against uncomplicated fibrositis and in favour of articular disease.

The differential diagnosis in favour of periarticular fibrositis is made on the persistence with which direct and indirect evidence of intra-articular disease remains absent. When objective changes, systemic manifestations, and alterations in laboratory findings known to be relatively consistent in cases of atrophic arthritis remain persistently absent, the arthralgia, stiffness, and articular thickening can be with no little confidence ascribed to periarticular fibrositis and the fear of impending arthritis can be laid aside.

FIBROSITIS AND INFECTION

By DOUGLAS H. COLLINS

Definition of Fibrositis

It is traditional and etymologically correct to define fibrositis as an inflammation of fibrous tissue; but this is an inadequate definition of the clinical state to which the name applies. It is not yet established that inflammation is the only process, or fibrous tissue the only tissue, concerned in the pathogenesis of the disease. Fibrositis must therefore be defined afresh in clinical terms. It may then be clear that the subsequent discussion concerns a familiar clinical condition without assuming any uniform and established pathology for it.

Fibrositis, then, is an acute or chronic painful state of the subcutaneous tissues, muscles, fasciæ, ligaments or tendons, arising independently of gross anatomical disease from which pain might be referred. The pain of fibrositis has the more or less uniform quality which Lewis[1] attributes to all pains arising from deep somatic structures, irrespective of the nature of their stimulus. It is felt both locally at certain tender points and referred according to segmental distribution (Kellgren[2]). The painful state of fibrositis is sometimes, but not invariably, associated with localised indurations or nodules palpable through the skin, and these frequently coincide with points of maximum tenderness. These so-called fibrositic nodules can sometimes be dispersed by massage and, if they are the focal points from which the pain originates, relief and even permanent cure of the symptoms can often be obtained by infiltrating them with local anæsthetic.

General Ætiology of Fibrositis

Fibrositis is usually classed as a rheumatic disease, and the numerous and varied ætiological factors attributed to other rheumatic diseases have also been assigned to it—*e.g.*, climate, occupation, trauma, endocrine disturbances, the "rheumatic diathesis", psychoneurotic states, and infection. The last only will be considered in detail here. There are good grounds for

believing that a high proportion of cases diagnosed by practitioners as fibrositis have an obviously non-infective ætiology. Amongst these may be mentioned—(*a*) the painful states caused by abnormal strains on ligaments and tendons, including the late result of acute traumatic sprains and the painful conditions of muscles caused by unwonted athletic exercise, fatigue, or faulty posture; (*b*) the painful states of the subcutaneous tissues in endocrine disease, seen in an exaggerated form in Dercum's disease; and (*c*) the varieties of muscular cramp resulting from local vascular disturbances. The clinical differentiation of these variously caused somatic painful states is often difficult. The doctor must, however, be aware of their variety and view the condition of fibrositis eclectically, not as a specific disease entity but as a common manifestation of a diversity of underlying conditions.

A precise ætiology cannot be ascribed to all clinical cases of fibrositis. Many cases are attributed to "infection," by which is generally understood the absorption of bacteria or bacterial toxic products from foci of bacterial growth in teeth, tonsils, gut, or elsewhere. Some practitioners recognise a state of fibrositis to co-exist with the more clearly defined diseases of rheumatic fever and rheumatoid arthritis. This should be regarded as a secondary fibrositis and a part of the major disease. It is not an argument in favour of fibrositis in general being one of many manifestations of a single comprehensive specific rheumatism. The cases of fibrositis which never show any signs of articular or cardiac disease are far more numerous. There is, however, a form of fibrositis which principally affects the tissues in the region of the joint, but it appears that this can be distinguished from actual arthritis (Slocumb[3]).

The conditions known as "muscular rheumatism" and "non-articular rheumatism" are included within the term "fibrositis" in my discussion. I shall make no special mention of neuritis. The neurologists have sufficiently defined certain specific forms of interstitial neuritis, and recent information about lesions of the intervertebral discs and of the ligamentum flavum is removing more and more cases of sciatica from the fibrositic category.

We have already seen that there are numerous cases which, for lack of a better word, can be diagnosed as fibrositis and which we now know to have a non-infective basis. The object of my discussion now is to consider whether infective causes can be

definitely attributed to any of the remaining cases. This I propose to do by examining the evidence under three heads: (1) morbid anatomy, (2) clinical pathology, and (3) analogy with known infective states.

1. *Morbid Anatomy.*—Our information on this subject is meagre. The disease is not fatal, and it is not recognisable in the post-mortem room. Biopsy material alone is available, but patients are reluctant to allow biopsies when their physicians cannot promise that the procedure will be beneficial.

The observations of Stockman[4] have been quoted over and over again in books and papers and have been generally accepted without criticism. Stockman excised and examined a number of fibrositic indurations. He described the microscopical changes as inflammatory hyperplasia of connective tissue which contained fibroblasts, a greater or lesser sero-fibrinous exudation, but no leucocytes. All cultures were sterile. " In view of our present knowledge," Stockman wrote, " it seems at least likely that these local fibroses are due to small colonies of microbes invading the tissues and causing a reaction which comparatively rapidly destroys the invaders. The organisms are non-pyogenic . . . there is no softening of the tissues and no pus formation, but instead they leave behind a small patch of inflamed fibrous tissue which persists and spreads." He went on to state that the exacerbations and relapses of the condition suggest a chronic general infection by an organism of attenuated virulence with " irritation from its toxin ". But he also stated that the frequent exacerbations and tendency to spread might be due to " irritating causes other than microbic, such as constantly recurring exposure to cold and damp ". Stockman's material is almost unique and consequently of great importance. I have examined some of it myself, but I am convinced that no deductions whatever concerning the causation of the fibrositic nodule can be drawn. The illustrations in Stockman's book are not impressive. Five of them show scarcely more variation in fibrous tissue structure than can be encountered normally in different situations in the body. The sixth depicts a fibrosed and re-canaliculised thrombotic vessel. In my view, Stockman's most important findings were negative ones—*i.e.*, the absence of leucocytic infiltration of the fibrositic nodules and the sterility of the cultures prepared from them.

In a later chapter Stockman considered panniculitis—a

painful nodular condition of the subcutaneous mesenchymal tissues which most physicians regard as a fibrositis. Here again the histological changes so nearly resembled normal anatomical variations that no important deductions as to ætiology can be made from them. Stockman noted that the painful subcutaneous nodules often resembled encapsulated lipomata; but he distinguished between the rheumatic condition and adiposis dolorosa resulting from disease of the pituitary gland. To me it seems quite possible, in view of the age grouping and general nutritional state of these patients, that minor endocrine disturbances may play a part in the so-called rheumatic panniculitis.

My own experience in the histology of biopsy tissue from cases of fibrositis may be recorded here. It is a limited and somewhat unsatisfactory experience, but so little is known of the subject that any information at all may be of value. I have examined upwards of fifty subcutaneous nodules from various forms of rheumatic disease. Of these, seven relate to the subject under discussion. They were kindly removed for me by Mr. T. V. Pearce, surgeon to the Harrogate Royal Bath Hospital and the Harrogate General Infirmary, and all except those in Cases 6 and 7 had been diagnosed by experienced physicians as "typical fibrositic nodules".

Case 1.—Female aged forty-seven. Chronic pain in the small of the back and right buttock following hysterectomy four years previously. Behind the right sacro-iliac joint "a massive fibrositis" was palpable. One large and a few small nodules could be rolled under the examiner's finger. They were tender. Their removal was followed by an improvement in the local symptoms.

Pathology.—The tissue consisted of irregular lobulated fatty masses enclosed in a fibrous capsule and lying in the general subcutaneous fat. Microscopically they resembled simple lipomata and showed no evidence of inflammatory change.

Case 2.—Female aged fifty. Pain in left buttock and down left leg for four years. "Large fibrositic masses in the lumbar and gluteal muscles or subcutaneous tissue." No X-ray abnormalities. A nodule was removed from the left buttock. The pain down the leg was not relieved.

Pathology.—Adipose tissue with some fibrous encapsulation. Probably a simple lipoma. The tissue is relatively avascular. No inflammatory changes.

Case 3.—Female aged twenty-five. "Fibrositic nodules" from the posterior sacro-iliac region.

Pathology.—Lobulated adipose tissue with no inflammatory changes.

Case 4.—Female aged thirty. Diagnosed as fibrositis by an experienced physician. A "fibrositic nodule" was removed from amongst the flexor muscles of the left forearm.

Pathology.—A firm white nodule measuring about 1 cm. in diameter, which proved on microscopical examination to be a typical neurofibroma. The case was thereupon diagnosed correctly as von Recklinghausen's disease.

CASE 5.—Female aged forty-two. Rheumatoid arthritis affecting many joints for five years. General condition much improved and sedimentation rate returned to normal. Numerous tender and painful subcutaneous nodules gave rise to the diagnosis of fibrositis secondary to the arthritis. A small nodule was removed from the plantar fasciæ beneath each heel.

Pathology.—Tough fibrous nodules which on section showed the typical structure of the subcutaneous nodules of rheumatoid arthritis, which I have described in detail elsewhere.[5] It would seem misleading to consider this fibrositic state as merely secondary to the arthritis. It is an integral part of the major disease.

CASE 6.—Female aged twenty-five, an ardent and persistent worshipper. A small hard and painful nodule overlay the left patella tendon and interfered with kneeling. The nodule when removed measured $1 \times 1 \times 1{\cdot}5$ cm.

Pathology.—The section showed an irregular mass of fibrous tissue lying in fat. The tissue was considerably vascular save in the central degenerated area. Many of the bloodvessels were cuffed by lymphocytes, and small lymphorrhages were also seen elsewhere. There were no polymorphs or plasma cells. Some of the collagen showed hyaline change and towards the centre of the tissue there were areas of loosely reticulated " fibrinoid " or " mucoid " degeneration of the fibrous tissue. There was no intense fibroblastic reaction around these areas.

This patient's nodule was never diagnosed as fibrositic. It was thought to be traumatic in origin, and the microscopical appearances are consistent with this view. The nodule, however, showed many of the structural changes which have been repeatedly quoted as being characteristic of fibrositis. The case is mentioned as a warning against the careless assumption of infection as the sole cause of such changes.

CASE 7.—Male aged eighteen, a student who for six months complained of a snapping middle finger. A small nodule, less than 1 cm. in diameter, was enucleated from the fibres of the deep flexor tendon. It gave rise to the symptoms by catching as it slipped through the fork of the flexor digitorum sublimis tendon.

Pathology.—The bulk of the nodule was myxomatous fibrous tissue irregularly spattered with fibroblasts and thin-walled capillaries. Infiltrating leucocytes were very rare. Expert histologists to whom I showed the sections failed to agree as to whether the tissue was granulomatous or neoplastic. This condition is unlikely to be called rheumatic, though it might be thought fibrositic and possibly related to the tendinous fibrositis of which Dupuytren's contracture may be a type. An infective ætiology was unproved in this case. The case further shows how difficult it is to decide upon the ætiology from the pathological appearances of even so well localised a lesion of fibrous tissue.

Of these seven cases, the first three examinations confirm Stockman's findings in panniculitis. They do not help us to come to any decision regarding ætiology. The complete absence

of inflammatory changes does not suggest an infective origin. The degree of fibrosis seen around the fatty masses was no more than that which occurs around simple lipomata. It did not amount to anything suggesting post-inflammatory cicatrisation. Some authors have described interstitial changes in the nerve-twigs in these nodules and thereby explain their painfulness. I was unable to satisfy myself on this point.

Case 4 was a straightforward and understandable misdiagnosis. It is described merely to show how wary the physician must be to exclude certain already well-defined conditions from the fibrositic category. Case 5 is intended to show that the "secondary" fibrositis accompanying rheumatoid arthritis may be an integral part of the major disease. Case 6 demonstrates that purely traumatic causes may explain some of the histological changes of fibrous tissue which have been thought essentially infective inflammations. Case 7 is a local condition of tendinous fibrous tissue of unknown ætiology.

The 1933 B.M.A. Committee on arthritis and allied conditions[6] describes the fibrositic nodule as "formed by blocking of the lymphatics followed by a localised leucocytic infiltration, and this, in turn, by fibrosis and scar formation". The description is not very enlightening. It is hard to understand and it might apply almost as well to a surgical cicatrix. The Committee goes on to say, however, that "there is no record of a recovery of micro-organisms from fibrositic nodules".

Many writers have regarded the painful nodules as muscular indurations. The small acutely tender lumps that can be felt within a few hours of the onset of some fibrositic pains do, indeed, suggest local spastic contractions of muscle, and these, moreover, can often be dispelled quickly by firm massage. Gräff[7] could find no histological changes in any biopsy material from cases of muscular rheumatism, apart from the specific Aschoff nodes, which he was able to find in voluntary as well as in cardiac muscle in acute rheumatic fever.

Fischer[8] describes as muscular rheumatism the same condition which we include under the heading of fibrositis, and considers it to be most frequently secondary to diseases of the joints and of the vertebral column. This he calls secondary muscular rheumatism. Primary muscular rheumatism Fischer divides into myositis (inflammatory) and myalgia (non-inflammatory). The causes he gives for inflammatory myositis are all specific infections

capable of manifesting themselves in other ways than subjectively felt pains—*e.g.*, septicæmia, trichiniasis, gonorrhœa, and rheumatic fever. Non-inflammatory myalgia he attributes to hypothetical changes in the physico-chemical state of the muscles, and these present no morbid anatomical features.

A great difficulty in assessing the results of pathological examinations is the fact that the tissues which are affected in fibrositis are not highly differentiated and therefore their modes of reaction to various noxious agents are limited in type. It seems unlikely, on this ground, that further examinations of the morbid anatomy of the fibrositic lesions will add much to our knowledge of their cause. It is for this reason, too, that I regard the experimental production of lesions which might bear some resemblance to those of fibrositis as not decisive. Gordon[9], by infecting rabbits with an attenuated variola virus, alone and in combination with hæmolytic streptococci, reproduced lesions of the periarticular tissues which he correctly termed "fibrositic", and sought to liken them to the lesions of human rheumatic fibrositis. The lesions that resulted from these experiments seem to me to be more clearly inflammatory than any yet reported as occurring in human fibrositis. This work of Gordon's is of great value in showing the synergic effects of a virus with streptococci and the manner in which the streptococci disappeared from the lesions when combined with the virus. The almost insuperable difficulty in experimental work directed towards the elucidation of fibrositis is the fact that in man fibrositis is essentially a subjective symptom which may or may not be associated with objective signs. The morbid anatomy of the condition in man remains so vague that its comparison with lesions produced experimentally in animals can never be free from doubt. Thus Stockman, to whom Gordon referred his sections, felt bound to say that though the rabbit lesions were "similar to those of rheumatic fibrositis in man" similar changes could be produced by other agencies—*e.g.*, trauma.

Some information might be gained from a comparison of common fibrositis with gonorrhœal fibrositis, but I have been unable to discover any details of the morbid anatomy of the latter beyond the bald statement in a textbook of medicine[10] with reference to the painful heel of gonorrhœa that "organisms have been found in this situation".

Studies in morbid anatomy seem to me to prove that frank

bacterial infection plays no part in ordinary fibrositis. Whether the lesions, such as they are, result from the action of bacterial toxins remains speculative.

2. *Clinical Pathology.*—The 1933 B.M.A. Committee on arthritis[6] quotes "septic foci, particularly in connexion with the teeth, tonsils, gall-bladder, and prostate", as some of the causes of fibrositis. This view was doubtless formulated as a result of the impression common among clinicians that focal sepsis bears an ætiological relationship to fibrositis. The view is not very well supported by the available statistical evidence, but it remains widely held by numerous physicians with a wide experience of the disease, and the fact that it is so held constitutes a powerful argument for its validity. The Ministry of Health 1924 Report[11] considered the incidence in the insured population of muscular rheumatism, lumbago, and sciatica. None of these conditions was significantly associated with tonsillar sepsis. Dental sepsis, on the other hand, was found to be very frequent amongst sufferers from these diseases, but, as it was also frequent amongst the population at risk but unaffected by rheumatism, it could not be regarded as of proved ætiological significance. A strong claim to this was, however, made in the report. But there was also a correlation between the frequency of dental sepsis and increasing age. Lumbago and sciatica, which in the light of more recent knowledge appear to owe more to mechanical than to infective causes, were also found in this survey to be associated with dental sepsis as frequently as was muscular rheumatism. Last year Vaizey and Clark-Kennedy[12] published a careful review of the relationship between dental sepsis and conditions of chronic ill-health, including rheumatism. They concluded that all the evidence of a causal relationship between dental sepsis and anæmia, dyspepsia, and rheumatism was equivocal and that serious criticism could be levelled against the bacteriological, statistical, and therapeutic data which have so far been produced to support such a causal relationship.

Laboratory examinations of the blood in fibrositis furnish no proof of the condition being infective. Secondary anæmia is uncommon.[13] The sedimentation rate is generally normal. There is no leucocytosis nor any marked shift to the left of the neutrophil nuclear count. Levinthal[14] found that agglutination and absorption tests with a group A hæmolytic streptococcus were negative in 73 per cent. of sera from cases of fibrositis, whereas

they were positive in 72 per cent. of cases of rheumatoid arthritis.

Sufferers from fibrositis may show signs of fatigue from persistent pain, but they seldom appear toxic or lose weight. Their generally healthy aspect is often in marked contrast to their miserable story.

No one who has seen many cases of fibrositis can deny that occasionally successful eradication of a septic focus is followed by relief of symptoms or that a painful fibrositic state sometimes follows the development of a focal infection. Focal infections which appear to bear a causal relation with fibrositis seem to me to fall into two classes. Examples of each will emphasise the necessity for distinguishing between them.

The first example is that of a young man who develops tonsillitis and continues his work for a day or two until waking up one morning he finds himself almost unable to get out of bed on account of severe pains in the back and limbs. The doctor is called and diagnoses acute muscular rheumatism or fibrositis and prescribes aspirin and gargles. The aspirin mitigates the pain. The tonsillitis subsides in its own good time. Severe fibrositic pain persists perhaps for several days, but as the sore throat gets better so does the fibrositis and the patient ultimately recovers completely. The doctor naturally and, I believe, quite correctly links the tonsillitis to the fibrositis, and he may later advise tonsillectomy " to prevent a recurrence ". In such a case the sore throat has been a hæmolytic streptococcal infection, and the acute fibrositis a manifestation either of a transient septicæmia or of an acute bacterial toxæmia and can be compared with the acute painful states occurring in other septicæmias. Tonsillectomy will prevent a recurrence only if, by this measure, the patient escapes a future streptococcal angina. It does not follow from this example of an acute fibrositis clearly caused by infection that all other fibrositis is so caused.

The second example of focal infection apparently related to fibrositis is the case of an elderly multiparous woman who for years has suffered from lumbago and a vaginal discharge. She is found to have a chronic cervicitis, and this is appropriately treated. As the focal infection is cured the lumbago disappears. The case makes an impression on the physician's mind of fibrositis relieved by treatment of a septic focus. The truth is that this lumbago is not fibrositis; it is referred pain.

It was discovered some years ago that local anæsthetic injected into and around a sprained ligament not only relieved pain but also prevented reactionary œdema and promoted recovery of function. Leriche,[15] the pioneer in this field, believed that the local anæsthetic, by its effect on the sympathetic nerves, arrested inflammatory reaction to injury. Local anæsthetic can relieve and often permanently cure the symptoms of fibrositis if the right points of maximum tenderness are injected. The fact seems to indicate that no bacterial irritant persists in these tissues. Nor is it likely that the injection of small amounts of anæsthetic solution into one or two areas of subcutaneous tissue prevents all future manifestations of a condition caused by the continual absorption of "toxins" from a septic focus. The success of this form of treatment in so many cases of fibrositis strikes me as a strong argument against the focal infection theory. It may also be noted that some formerly ardent advocates of focal infection and vaccine therapy are to-day using procaine injections extensively in their treatment of fibrositis.

3. *Analogy with Painful States in Known Infections.*—Pain in the muscles, joints, and fasciæ frequently occurs in the early stages of many acute febrile diseases—*e.g.*, typhoid fever, cerebro-spinal fever, influenza, scarlatina, relapsing fever, leptospirosis, smallpox, and typhus. Such pain may last through the whole course of other diseases—*e.g.*, brucellosis, trench fever, and dengue. The somatic pains, in the bacterial and protozoal infections, are contemporary with the septicæmic phase of the disease. This is possibly also the case in the rickettsial and virus diseases. The acute fibrositis in streptococcal angina may be compared with these examples. Generalised skeletal pains seem to be a frequent concomitant of septicæmia without distinction as to the invading micro-organism.

There is an acute febrile disease, not common in the British Isles, the major symptom of which is muscular and tendinous pain. Varieties of it have been named acute epidemic myalgia, "the devil's grip," or Bornholm disease. Its precise cause is unknown. It is communicable. Recovery is complete. It seems to bear no relation to common fibrositis.

Non-articular skeletal pains are now a well recognised symptom of acute rheumatism (rheumatic fever). Anatomical lesions resembling the Aschoff nodes of the myocardium have been found in the voluntary muscles by some (Gräff[7]), but others believe

that the toxins either of a streptococcus or of an unidentified specific virus cause these pains. Healey[16] noted that transient joint pains occurred during active immunisation with scarlet fever streptococcus toxin. A high proportion of the persons who developed these pains had a history of previous joint pains suggestive of rheumatic infection. The sensitivity of rheumatic fever subjects to sterile streptococcal products has also been shown by many others (*e.g.*, Green[17]). The muscular pains of acute rheumatism are usually excluded from the category of fibrositis. The average patient with fibrositis shows no evidence of previous specific rheumatic infection, and it seems wiser to consider the two diseases as being entirely distinct.

Pyæmia, glanders, trichiniasis, and dermato-myositis cause focal inflammatory lesions of fibrous tissue and muscle. Histologically the lesions are quite different from those described in fibrositis.

From this short review it will be seen that painful states of muscle and connective tissue may result from bacterial septicæmia or toxæmia, and focal anatomical lesions from pyæmia and certain specific infections. But the general features of these diseases are so unlike those of common fibrositis that the analogy does not force the conclusion that the latter condition is also necessarily infective in origin.

Summary

The term fibrositis covers in common usage the ætiologically undefined conditions named myositis, myalgia, non-articular rheumatism, panniculitis, and periarthritis.

Fibrositis is a common clinical condition of vague pathology. It should be defined in clinical not in pathological terms. This I have attempted to do (p. 114).

A high proportion of cases of fibrositis have an obviously non-infective ætiology.

Fibrositis occurring in the course of rheumatic fever and rheumatoid arthritis has, on occasion, been proved to be associated with nodules in subcutaneous tissue or voluntary muscles characteristic of the major disease. No similar lesions have been described in fibrositis arising independently of these diseases. There is no reason, therefore, to regard all cases of fibrositis as being manifestations of a specific rheumatic infection.

The known morbid anatomy of fibrositis has been critically

discussed and some new examinations of biopsy tissue have been recorded. Evidence of inflammation caused by infection in the fibrositic nodule is very slender. Leucocytic infiltration is generally absent. Cultures are always sterile. Reactions of connective tissues to various noxious agents are limited in type. No decision regarding the ætiology of the fibrositic nodule can be made on the available pathological data.

Animal experiments are unlikely to elucidate the causes of human fibrositis, which is essentially a subjective phenomenon. It is also often difficult to distinguish between fibrous tissue lesions arising from different causes.

Laboratory examinations of the blood in fibrositis lend no support to the view that the condition is generally infective.

Only in rare instances can it be considered proved that focal infection is a cause of fibrositis.

Acute focal infection, especially hæmolytic streptococcal tonsillitis, may cause acute fibrositis. Body pains, resembling acute fibrositis, also occur during the septicæmic or invasion phases of many specific infective fevers.

Chronic focal infection, particularly of the pelvic organs, may give rise to chronic referred pain. Disappearance of this pain on treatment of the deep-seated infection may be misinterpreted as cure of fibrositis by removal of a septic focus.

General Conclusion

Acute septicæmic and toxæmic infections can cause acute fibrositis. Chronic infections of viscera can cause chronic referred pain. Rheumatic fever and rheumatoid arthritis, both probably infective diseases, can cause specific varieties of fibrositis. Painful nodular lesions of muscular and connective tissue develop in certain specific infections—*e.g.*, glanders and trichiniasis. Gonorrhœal fibrositis is recognised. Apart from these instances there is no sound evidence that fibrositis originates in infection. Other ætiological factors are at least as likely to be concerned in the majority of cases of fibrositis.

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LABORATORY FINDINGS IN FIBROSITIS

By JOSEPH RACE

(*From the Laboratory of the Devonshire Royal Hospital, Buxton.*)

In this review of the biochemical characteristics found in fibrositic conditions the scope will include fibrositis due to trauma and occupation, "gouty fibrositis", the periarticular type in which the manifestations are in the periarticular tissues without involvement of the joints themselves, and some cases of sciatica in which the nerve symptoms are secondary to fibrositis.

Most of the data are obtained from routine examinations made in the hospital laboratories during the last five years, but the numbers of the various types bear no relation to the incidence of the types admitted for treatment; further, as the hospital serves a large industrial community, the cases of occupational or industrial fibrositis naturally preponderate.

In general the laboratory findings in the type of disease under consideration show no deviations from the normal, but these negative findings are by no means without value, since they serve to exclude those diseases which give abnormal results.

One of the most useful laboratory tests in all rheumatic diseases is the determination of the suspension stability of the erythrocytes (sedimentation rate), and especially so if it is combined with the determination of the cell volume as suggested by Collins *et al.*[1] This method not only gives the suspension stability (S.S.), but also detects an excess or deficiency of red cell volume, which is correlated with the amount of hæmoglobin present, and any marked deviations from the normal in the number of leucocytes present. The colour of the plasma also yields useful information, an excess being often due to an excess of bilirubin, while a deficiency is usually associated with a lack of carotinoid bodies, one of which is provitamin A.

During the past five years the S.S. has been determined on some 620 cases of fibrositis of all types, and the results are shown in Figs. 1 and 2, which relate to the males and females respectively. This division was made because the normal values for the two sexes are different owing to the lower average red cell counts in

females, which is reflected in a lower cell volume and reduces the S.S. by approximately 5 per cent.

In Fig. 1 it will be seen that, although a very large proportion

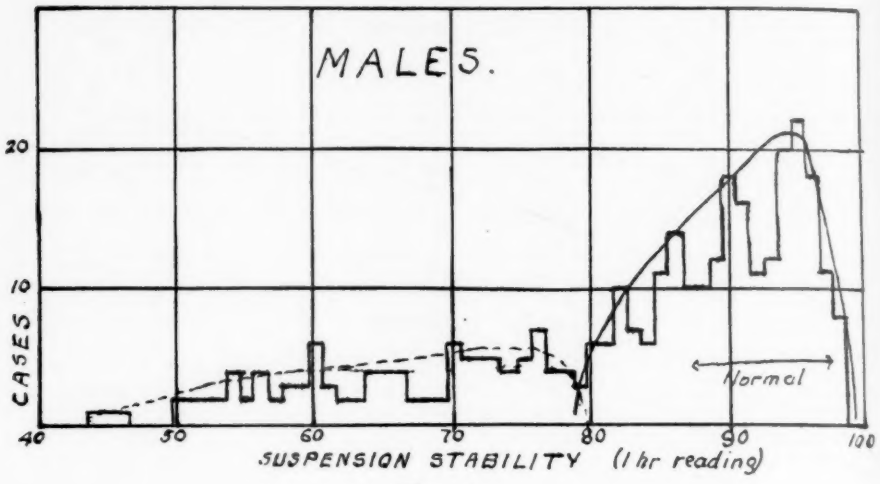

FIG. 1.

show but little or no deviation from the normal, there is an appreciable number in which the stabilities are so lowered as to resemble those found in rheumatoid arthritis. In Fig. 2 the cases with a

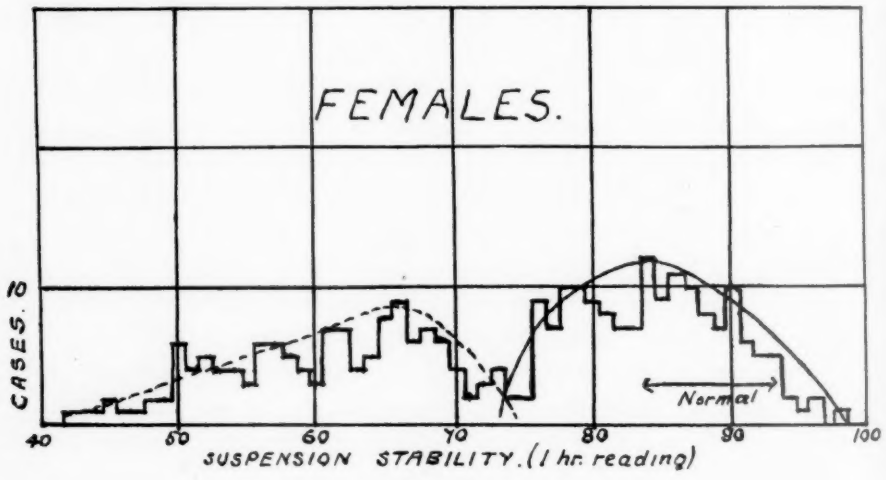

FIG. 2.

low stability are greater in number and seem to form a definite group, although it must not be overlooked that the separation into two groups is somewhat accentuated by the relatively smaller incidence of the industrial type found in females.

In both sexes the spread of the normal groups is undoubtedly extended by the cell volume factor, and if the readings had been corrected to a standard cell volume these variations from the normal would have been very materially reduced and the sex effect would probably disappear.

An analysis of these cases demonstrated that whilst several factors were apparently involved in the production of the low stability, its association with the periarticular type of fibrositis was well defined, and the following table shows that it was low —in an appreciable number very low—in over 80 per cent. of the cases. Many of these results are appreciably more abnormal than any reported by Slocumb,[2] but the tendency towards lower stabilities with increase of the duration of the symptoms is also quite definite in this series.

Suspension Stability.	*Periarticular Fibrositis.*			*Gouty Fibrositis.*		
	Males.	*Females.*	*Total.*	*Males.*	*Females.*	*Total.*
41-50 ..	2	1	3	1	1	2
51-60 ..	6	7	13	2	3	5
61-70 ..	5	5	10	3	1	4
71-80 ..	4	4	8	4	2	6
81-90 ..	3	4	7	5	1	6
91-100 ..	0	1	1	5	1	6
	20	22	42	20	9	29

In " gouty fibrositis ", for which the results are shown in the same table, a few low stabilities are found but, as in the articular type, the results are very variable. A further contrasting feature between these two types lies in their response to treatment, the periarticular one being much less responsive than the gouty.

In seventeen patients diagnosed as sciatica secondary to fibrositis the S.S. was normal, or practically so, in all cases. In none was there any muscle wasting which was noted previously[3] and is often associated with a marked reduction of the S.S.

The results with the fibrositis group as a whole are very similar to those reported in 1929,[3] but it is now recognised that the lower results generally found in females as compared with males are due largely to the lower red cell volumes natural to the female sex. This physiological difference between the sexes is generally accepted, and according to the data of Williamson,[4] who examined 919 samples by spectrophotometric methods, the variation commences before the first decade of life, reaches a maximum in early adult life, and continues to the end of the seventh decade.

It has been suggested that this physiological difference between the sexes is not confined to the red cells, and that similar variations are to be found in the plasma constituents.[5]

Uric Acid.—The existence of "gouty fibrositis" as a distinct syndrome is still controversial among those interested in rheumatic and allied diseases, and whilst it is commonly recognised in this country, an attitude of extreme scepticism prevails in North America, where one prominent rheumatologist has expressed the view that he is unable to accept the "gouty" qualification until one single case can be produced showing a uratic tophus. A tophus is essentially a uratic deposit in the superficial tissues, and consequently a visible one, but there is no obvious reason they should not occur in the deeper tissues. The use of unformolised sections from nodules in cases of "gouty fibrositis" may possibly lead to a solution of the problem. It is a matter for discussion whether the gross evidence of tophi is a more reliable index of gout than a high level of uric acid in the blood associated with clinical characteristics of the disease.

The uric acid content of whole blood from cases of "gouty fibrositis" and from a number of non-gouty fibrositic ones is given in the following table:

	Males.		*Females.*	
	No.	*Average Uric Acid (Mg. per Cent.).*	*No.*	*Average Uric Acid (Mg. per Cent.).*
Gouty fibrositis ..	43	4·79	16	4·35
Non-gouty fibrositis ..	35	3·54	24	3·27

In a review of the laboratory findings in this hospital for the period 1923-1927[5] a much higher average was reported for both male and female cases of fibrositis, but the difference is largely due to changes in the method employed for the estimation of uric acid. The earliest quantitative method included the "combined uric acid" of French workers, a substance later proved to be thioneine and found almost exclusively in the red cells. In 1930[6] Folin developed a uric acid reagent of much greater specificity, and the results given in this contribution were obtained with this reagent. Many workers on the Continent and in North America have eliminated the error due to thioneine by employing blood plasma, but it should be remembered that the red cells contain comparatively little uric acid, so that the plasma figures are much greater than those for whole blood.

The uric acid results given in the table show a significant

difference between the "gouty fibrositis" and non-gouty groups, and whilst an abnormal uric acid content is certainly not a specific indication of gout, it does suggest that the fibrositis may have a gouty basis.

VITAMINS.—In the predecessor of this journal[7] the writer reviewed the relationship of vitamins to rheumatic diseases and included a number of data as to the amounts of vitamins A and C found in the blood of fibrositic patients. Since then laboratory methods have advanced to such a degree that it has been possible to extend the investigation to the determination of vitamin B_1.

The process used was essentially that of Wang and Harris,[8] in which the amount of the vitamin excreted in the urine is estimated after the removal of the preformed thiochrome by extraction with *iso*-butanol. A small amount of the extracted sample is oxidised with potassium ferricyanide in alkaline solution and the thiochrome extracted with *iso*-butanol, which is then washed with water and compared visually against a standard solution of pure vitamin B_1 under an ultraviolet lamp. The process enables very small amounts of the vitamin to be estimated with a reasonable degree of accuracy.

A twenty-four-hour sample of urine is collected, and bacterial development must be prevented by the addition of a suitable preservative such as toluene, for many types of micro-organisms are able to produce appreciable amounts of the vitamin. According to Professor L. J. Harris (verbal communication) it is also essential that the patient should not be treated with aspirin or similar preparations. The writer has found that cases of rheumatoid arthritis under treatment with myocrysin occasionally excrete comparatively large quantities—over 1,000 international units (I.U.) per litre—of some substance that reacts in the test exactly like vitamin B_1.

The results expressed as I.U. per diem are shown below together with some normals for comparison:

Normals: 90, 75, 80, 68, 87, 45, 60, 108. After injections of benerva 175.

Fibrositis: 19, 28, 30, 26, 29, 19, 32, 38, 34, 28, 36, 25, 28, 37, 23. After taking betaxin 190.

Wang and Harris,[8] using eleven normal subjects consisting of laboratory assistants and research workers, found a minimum excretion of 30 I.U. per diem and a maximum of 150 I.U., the

majority being between 50 and 80 I.U. In some nerve diseases they obtained much lower results: subacute combined degeneration, 15, 16; disseminated sclerosis, 16; peripheral neuritis, 26, 0, 0; anorexia nervosa, 19, 20; and in simple anorexia, 29, 16, the results being obtained at various dates on the same patient. They remark: "The subnormal values found . . . may presumably be attributed to 'conditioned' deficiency (or partial deficiency) arising from faulty absorption or utilisation or restricted diet."

The writer has found that other types of rheumatic patients excrete vitamin B_1 in amounts somewhat similar to those found in the urine of fibrositic ones, and the deficiency must therefore be mainly attributed to some common factor, which in the patients admitted to this hospital is probably a dietary deficiency due to the economic conditions obtaining in the class from which they are chiefly drawn.

In previous work[7] the vitamin C content of the blood was found to vary with the economic strata of the patients, and in view of the deficiencies of vitamins A and C found in the patients admitted to this hospital a similar deficiency of B_1 was considered probable.

A number of the vitamin B_1 values found in the cases of fibrositis were no greater than some of those found by Wang and Harris in definite neurological diseases, but they were not accompanied by any symptoms attributable to vitamin B_1 avitaminosis. Many of the patients were males, apparently healthy apart from localised fibrositis, who had been capable of doing a full day's work in heavy occupations. The vitamin deficiencies in such cases suggest that there is a very considerable margin between pathological and optimal levels of vitamin content. Immediately above the pathological level is evidently a suboptimal zone of considerable depth and of great importance from the point of view of national nutrition. It seems very desirable that vitamin studies should be made on a cross-section of the population of the country to determine the depth of this suboptimal zone, for it is not impossible that the present optima are extravagantly high and that what is now regarded as a "conditioned" deficiency really represents merely a low margin of safety. Whilst the importance of vitamins should be fully appreciated, there is a possibility that zeal has outstripped discretion, and that some of the present optimal levels represent

an abundance rather than sufficiency. Perhaps the war will provide a large-scale experiment in this direction.

INDOLE AND INDICAN.—In 1935 Forbes and Neal,[9] using improved methods for the estimation of indole and indican, examined urine samples from a number of rheumatic cases and reported considerable variations from the normal. Various types—infective arthritis (specific), rheumatoid arthritis, and osteo-arthritis—showed a markedly increased excretion of indole, whilst as regards indican the arthrides as a whole tended towards subnormal excretions, which was accentuated in the osteo type. A few high results were obtained in the rheumatoid type.

A series of cases in the Devonshire Royal Hospital has been studied by the methods used by Forbes and Neal, but the investigation has not yet reached the stage that warrants publication of the whole data. As regards fibrositis it will suffice to state that, although many cases show an increased urinary excretion of indole, the findings do not necessarily implicate the rheumatic condition. Little or no variation from the normal excretion of indican has been found, but the results definitely confirm the contention of Sharlet[10] that the usual figures (4 to 20 mg. per diem) found in text-books are much too low. His results for twenty-four-hour specimens varied from 40 to 150 mg., whilst Forbes and Neal[9] found an average of 73 mg. with a range from 30 to 165 mg.; the writer's results agree with these values.

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FIBROSITIS AMONG INDUSTRIAL WORKERS

By GERALD SLOT

A LARGE number of disability certificates are signed every week for industrial workers suffering from fibrositis. It has been noticed that some of these have been associated with their occupations. They can be classified:

1. *Fibrositis of Neck Muscles.*—This includes market porters, especially fish and vegetable porters, who carry heavy weights on their heads—the weight of the contents of their pile of baskets may reach between 50 and 100 pounds. Taxi drivers and lorry drivers are also represented in this group.

2. *Fibrositis of Arm and Hand Muscles.*—This group includes bus conductors, locomotive firemen, gardeners, and dustmen.

3. *Fibrositis of the Back and Gluteal Muscles.*—Sufferers from this form the largest group of occupational fibrositis. It includes bus drivers, miners, policemen, gardeners, milk roundsmen with heavy trolleys to push, tram drivers, iron workers, and stokers. Women engaged in lifting (shop and warehouse workers) are often among the group.

4. *Fibrositis of the Foot Muscles.*—This is a difficult group to classify, as foot defects—pes planus, etc.—should be separated. Arthritis of the feet is common, as also is referred pain from a sciatic lesion. Foot strain is a common association. The employments especially liable to this condition are shop girls who have to stand for long periods of time, recruits in the Army, and bus, lorry, and locomotive drivers.

The common factor in these varied conditions is muscle fatigue and trauma. The trauma may be slight but recurrent. The ætiology of these conditions is obscure, and because the condition is non-fatal, proof by biopsy can seldom be made. Other common factors are heat, humidity, and exposure to cold. The pathology may show small nodules under the skin—it is difficult to excise these as they slip away from under the forceps. On examination they are found to consist of small aggregations of round cells and fibrous tissue.

Though various workers have tried to grow organisms from

these nodules, results have been variable and unconvincing, and the author and his colleagues have never yet succeeded in growing an organism from a nodule removed either from a case of fibrositis or rheumatic fever.

Further changes include swelling and reddening of the fascia and a slight œdema of the surrounding tissue. Small punctate hæmorrhages—as if caused by the rupture of an attachment between fascia and muscle or fascia and skin—are sometimes seen. Pain in varying degrees is present and a slight rise of temperature is frequent. Trotter and Davis have explained the hyperæsthesia by assuming that there have been multiple minute injuries and that during the subsequent regeneration the nerve fibres growing from a central source fail to develop sheaths and neurilemma which insulate them from surrounding tissues. Head's explanation is that the hyperæsthesia is due to the liberation from the controlling and regulating influence of the touch fibres which may be affected by the inflammatory change. Some of these fibrositic nodules must be largely œdematous, because they disappear so rapidly, and the speed of their onset and disappearance makes one postulate an allergic mechanism of onset in some cases.

In industrial fibrositis, strains of muscles and ligaments are important predisposing causes. This is important, as workers exposed to similar conditions of work and climate so often suffer differently. In one hospital largely attended by transport workers it has been noticed that the omnibus workers, divided into the two groups of drivers and conductors, suffered differently. The drivers were frequently group 3 and complained of sciatica or lumbago, while the conductors were groups 2 and 4, suffering more from fibrositis of the hands and feet.

Common factors to all these sufferers is that they are cold-sensitive. They nearly always have bluish limbs and have frequently suffered from chilblains. They need hot-water bottles in their beds at night and are poor sea bathers.

The incidence of rheumatism in miners has been discussed by Buckley, who found fibrositis to be more common among miners than among moulders, glass blowers, furnacemen, and other workers exposed to high temperatures. He also stresses the greater incidence among outdoor workers subject to more exposed conditions than among indoor workers. Arising out of this it is interesting to note that fibrositis is not common among

postmen, who, although outdoor workers, maintain good circulation by constant walking, and I am informed that it is not an outstandingly important cause of ill health in the Army.

The occupational fibrositis of the neck type, group 1, usually starts with a feeling of discomfort in the neck muscles. Sometimes there is a fairly severe headache, located to the posterior part of the head, for a day or two. These neuralgic headaches frequently precede the actual fibrositic attack, and some patients never get beyond the headache stage, and diagnosis is then difficult. This type of headache is possibly due to an occipital fibrositis, and it disappears quickly on injection with procaine in the appropriate way. The pain then locates itself definitely in the complexus and trapezius muscles, and the patient will complain that he is unable to carry the same weight on his head and sometimes that he cannot move his head with former freedom. The pain is continuous, burning, and radiating through the neck. Movement intensifies it and the patient finds that lying flat is difficult, as the posture of the head extends the neck muscles. Why has market portering caused this ? It is due to an accumulation of small injuries to the muscles, of which the attachments are stretched and even ruptured. Small punctiform hæmorrhages occur in the muscle sheath. Sometimes grosser hæmorrhages, which can be seen under the skin, are present, and the further tension of the tissues increases the pain. These attacks sometimes come on acutely without any previous warning, and are then often precipitated by strain and cold.

What is the effect of cold ? In these patients it causes a local vasocongestion—the muscles are working hard and the products of muscle metabolism are not removed sufficiently quickly. This gives rise to cramp, especially in a loaded muscle. The part played by humidity may be that when fatigue products accumulate in a muscle, water is attracted and the muscle becomes larger. In a humid atmosphere the interchange is less readily effected. Occupations, therefore, in which strain of the neck muscles and exposure to cold and/or damp exist are likely to give rise to this group.

An obvious example of the part played by trauma is the fibrositis of taxi or lorry drivers, often seen in the shoulder region. Their driving-cab is exposed, and because they have the habit of opening the door by pushing the right shoulder against it, fibrositis is more common in the right than in the left

shoulder. They present a constant history—a grumbling pain followed by rigidity and a severe pain extending up the side of the neck and over the shoulder girdle. This prevents adequate arm movement, and if untreated lasts four to five days. On examination, apart from the restricted movement and tenderness of the trapezius and deltoid muscles, certain spots about the size of a sixpence can be identified. These are indurated and more tender than the surrounding area. In some cases actual nodules can be felt in the muscles.

Fibrositis of the arm muscles in gardeners, dustmen, and conductors presents the same picture of pain, usually in one arm, radiating down the arm to the wrist, the distribution not following any segment but involving the whole limb. In the "arm" suffering there is tenderness over the biceps and the flexor group rather more than in the extensor group. The condition is of course quite different from "writer's cramp," which from the combination of pain, spasm, loss of control, and tremor suggests the basal ganglia as the focal point of the breakdown which produces the disability. In all these craft palsies (telegraphist's cramp, gold-beater's cramp, milkman's cramp, etc.) the pain is always in the muscles and never in the joint. The factors again present in this group are strain, exposure, and trauma.

The third group is the largest. Cases of fibrositis of the lumbar and sacral muscles may be present merely as a pain in the back or as a severe lumbago or sometimes—as in a case recently seen—may be so acute as to make a differential diagnosis from a calculus in the urinary tract very difficult.

Patients may start with intense pain of a colic type which "doubles me up, doctor," and with great tenderness over the lumbar and abdominal muscles. Temperature and other manifestations of a feverish illness are often present. This type of fibrositis is commoner among drivers, and is as frequent among tram drivers, who stand, as among trolley and ordinary bus drivers, who sit. The onset is sometimes gradual, but more often sudden. The patient complains of a pain in the small of the back, and often also of pain in the back radiating down the back of the thigh and thence to the calf to be fixed in the heel. Pain is caused by extending the leg, and local hyperæsthesia is almost always present. Deep pressure over the posterior superior spine is painful and rotary movements of the spine are impossible. One case seen recently resembled an acute abdomen

in its dramatic onset and abdominal tenderness and rigidity. Repeated questioning produced a history of a recent soaking, and grumbling pains over the back on the day previous to examination, and thus led to the right diagnosis. These cases are difficult because they tend to recur and are completely incapacitating. Three weeks is a common period of illness, and one attack seems to predispose to further attacks. There is some difficulty in securing industrial conditions which afford relief.

Fibrositis of foot muscles must be distinguished from lesions of the foot itself. In many cases there is a pes planus, or unsuitable footwear may be the cause of strain. Arthritis of the feet is of course common. Some of these cases of so-called fibrositis of the feet have on investigation proved to be cases of intermittent claudication. We have had some seven or eight of these under treatment simultaneously and found no common industrial occupation—among the patients were two general labourers, a bus conductor, a tailor, and a typewriter mechanic.

In treatment, focal sepsis must be considered, although it is only a factor—and a small one in some cases. Certainly removal of carious teeth or tonsils does not cure the majority of the sufferers although it helps to improve the general condition. Other important aids to this end are fresh air, regular bowel movement, attention to the hypochlorhydria often present, by giving 10 m. of dilute hydrochloric acid three times a day, and treatment of the secondary anæmia, by the administration of iron with vitamins A and D. We have not found vitamins B and B_1 of any value, whether given by mouth or hypodermically, in these cases. General ultra-violet light is of value.

The removal of the industrial cause is often fraught with difficulty. Doctors should at times visit the workshops, factories, and garages to see for themselves the kind of work entailed. No one who has not driven a heavy lorry or bus on a dark winter night—especially under black-out conditions—realises the big physical and mental strain involved.

To give rest is essential. The patient is usually unable to change his occupation, and before his return to work the careful doctor will consider how, if the conditions of the job cannot be altered, those of the workman can. Advice to keep the driving-cab window closed, the use of an air cushion for long journeys, and the provision of efficient protection against weather where

the worker is exposed, is all of value. Advice on the wearing of warm underclothing and the adjustment of boots by wedging or other means must also be given. It is possible and desirable to improvise methods of home therapy, as the nature of the disease or the distance of the patient from a hospital may sometimes make travelling difficult. Expense is also a factor, and for those unable to walk it invariably means that a friend is required to accompany the patient. Add to this the loss of time in waiting and the subsequent going out into the cold, and one is sometimes driven to say that the result from attending a hospital or clinic may bring more harm from exposure and exhaustion than benefit from the fifteen minutes or so of therapy that may be given.

The cardinal medicament is heat, and preferably hot moist heat. A really hot bath (105° F.), in which $\frac{1}{4}$ pound or more of magnesium sulphate has been placed, is of value. The patient is instructed to soak in this and to try to move his painful limb or back *gently* under the water. The water should support the weight of the limb during this proceeding. An electric pad, associated with some analgesic such as lin. methyl. salicyl., is also useful. Failing this a hot-water bottle or kaolin poultice. Hot fuller's earth is good. A convenient method is to place 1 pound of the earth in a double saucepan, add about 5 ounces (or just sufficient to make a thick paste) of water, and heat until it is about 150° F. Then tip the paste out on to a piece of cellophane—large enough to cover a considerable part of the area affected—roll it flat with a rolling pin to the thickness of about $\frac{3}{4}$ inch, and tuck in the cellophane to make an envelope. Cut a window in the cellophane with a pair of scissors and place over the affected part covered by a piece of gauze. The cellophane and the earth can be used repeatedly. This method is of great value in home treatment.

For fibrositis of the hands it is helpful to fill rubber gloves with boiling water, run the water out and when fairly dry put on the hand, closing the wrist area with a rubber band. Contrast hot and cold baths for the feet are easily arranged at home, and are of value, especially if a rubefacient such as mustard is placed in the hot bath.

In the clinic the most useful methods are infra-red rays, gentle massage, and diathermy. For the continuous fibrositis galvanism is helpful, particularly if associated with salicylate

ionisation. One of the most valuable of all methods is the injection of 1 per cent. procaine into the muscles. About 5 to 10 c.c. can be injected in various painful areas at one time, and in some cases the relief of pain is almost immediate. The nodules should be transfixed by the needle and about ½ c.c. of procaine injected into them.

With sciatica I have had the best results from epidural therapy and manipulation.

Drugs are of secondary importance. There is no specific drug. Gold is contra-indicated in these cases. Analgesics such as veganin or aspirin are the most helpful. Twenty to thirty grains of Dover's powder at the beginning of an attack is useful, as is the use of salicylates in daily dram doses. In the few cases in which sulphonamide therapy was tried we noted no success. Iron and arsenic are useful if there is general anæmia.

Sleep is important. Barbitone soluble or phenobarbitone or amytal-aspirin at night will usually ensure it. We have not found vaccine therapy to be of any value.

Diet should be confined to light, simple foods. Plenty of fluid is needed.

Above all, a day or more in bed is of great value. Complete physical rest is important and a vital part of any scheme of the treatment. It must be remembered that many of the patients are over-tired and physically exhausted not only by their work and the constant recurrence of the conditions causing the disease, but also by their efforts to continue in the presence of fibrositis. The effort needed to do this is very great.

It is clear that this urgent problem must be tackled in the same way as tuberculosis, with efficient centres of treatment under skilled guidance. Various industrial and municipal schemes exist at Bermondsey and other places, but cannot be described here.

Always, however, there will be a place for home treatment and for improving conditions of work so that sufferers will not be so numerous.

THE ARTHRITIC SEQUELÆ OF PNEUMATIC DRILLING*

By W. S. C. COPEMAN, M.D., F.R.C.P.

The industries in which pneumatic tools are now employed are numerous and increasing. The chief amongst these are: mining, quarrying, road-making, shipbuilding, locomotive and other workshops, construction of all-metal aeroplanes, and shoe-making. The tools employed are pneumatic chisels, hammers, riveters, road drills, pounding and lasting machines.

Contrary to expectation, pneumatic tools have not been found to cause much occupational trouble.[1] The condition of "dead hand" is, however, a well-established vasomotor sequel of their prolonged use, and consists in a Raynaud-like syndrome showing tingling, numbness and coldness of the fingers, and in some cases of the whole hand. The report of the Chief Inspector of Factories for 1927 states that apart from the condition of "dead hand" there were few complaints about pains in the joints or muscles; and only one case of Dupuytren's contraction was observed. This is surprising in view of the nature of the work.[2]

There is no evidence that vibration by itself, without cold, can cause the vasomotor lesions.[3] Riveting machines, indeed, have a cold-air exhaust (which may be covered with ice) which is directed on to the hands of the workman. There is no doubt that it is this which brings on the attacks of spasm. The rate of vibration does not seem to be of great importance, but there is some evidence that the amplitude of the machine may play a part in determining the onset of this syndrome.

Small areas of decalcification of the bones of the carpus appear to be a later stage and were described by Brailsford (1934: "The Radiology of Bones and Joints," p. 27, London). Another change is apparent periosteal overgrowth on volar surfaces of shafts of phalanges. This sometimes occurs, however, in normal man (Kohler, A., 1931: "Roentgenology," p. 17, London).

Seyring describes three groups of quite distinct illness or

* A communication made to the Association of Physicians at their meeting in Birmingham, 1939.

injury which are incontestably related to the occupation of using a pneumatic drill:[4] (*a*) disturbances in bloodvessels; (*b*) affections of the muscles; and (*c*) injuries to joints.

Actual injuries of joints were first described by Holzmann in 1929.[5] They have generally occurred in miners, hewers, metal workers and riveters. In eight out of ten of his cases the right elbow was involved, the symptoms being pain and limitation of movements, especially extension. In one case detachment of a piece of bone occurred, and this had to be removed by operation. The duration of employment had been three years in one case, and in others six to ten years. The changes were described under such varying terms as "arthritis deformans," "myositis ossificans," "osteo-chondritis dissecans," "chondro-dermatitis," etc. It was largely on the basis of this report that the condition was included amongst diseases and injuries scheduled under the German Workmen's Compensation Act, 1929, in the following terms: "Disease of muscles, bones and joints from the use of pneumatic drills." Vasomotor disturbances are not included in this definition.

I have been privileged through the courtesy of the Home Office and also of Dr. McLaren[6] to examine two series of X-rays of men who had been using pneumatic tools for varying periods. I have also found several cases attending hospital. No evidence was found of changes of any sort under two years. It appeared, moreover, as would be expected from the way in which most workers grasp the machine, that where changes could be seen in the wrist they most often affected the radial side, the commonest lesion in my experience being a slight increase ("spiking") in the angles of the trapezium bone. This, however, appears to be symptomless, but when marked is sometimes reported as being an early osteo-arthritic change. The cysts mentioned above were not infrequently observed in these series.

Middleton[7] found localisation to vary, but, although the radial side was more often affected, the thumbs never suffered. X-rays seen appeared to show changes slightly more frequently on the radial side, suggesting that trauma from the employment was a factor in their production.

Moulonguet-Doleris showed some X-ray films of the elbows of workers with pneumatic tools at the Second Annual Meeting of the French League against Rheumatism in 1931 which demonstrated changes in these joints, and similar changes have also

been described subsequently by Mauclaire and Minet[8] and Sommer,[9] whilst Sigand and Terray[10] described a case in 1932 of occupational arthritis of the left wrist in a right-handed miner which they thought was due to the use of a pneumatic drill. This was, they stated, the first lesion of this nature to be reported. Junghanns[11] reported a similar case at considerable length in 1937, and illustrated his paper with an excellent X-ray showing "pseudo-arthrosis" of the navicular bone. Other cases have been reported by Hardgrove and Barker.[12]

W. C. Meiss[13] examined X-rays of 107 workers in Holland who had used pneumatic drills for one to twenty-five years. In twenty-four of these articular changes were recorded: osteophytic changes at head of radius and at the first carpo-metacarpal joint; also myositis ossificans on the anterior surface of the humerus at the origin of the brachialis anticus and at the insertion of the same muscle and of the triceps. Lesions were mostly limited to the right side in right-handed people, but in one case of a left-handed worker all these changes were on the left side. There was very little disability clinically. As a control series Meiss examined radiographically 100 workmen of various other occupations between the ages of forty-eight and sixty-five and found no osseous changes of this nature.

In 1934 F. W. A. Weber[14] described a case of a sixty-five-year-old man who had worked as a steel cleaner for fourteen years who developed ulnar nerve paralysis and muscle wasting and who was found radiographically to have a severe osteo-arthritis of the right elbow-joint. P. Rostock[16] in the same year mentioned a miner who showed arthritic changes in his right elbow after working with pneumatic drills for nine years. He was re-examined five and a half years later while still at work, and these changes were then found to be gross. Another worker who showed changes after four and a half years was taken off work three months after the examination, and one and a half years later the changes were found not to have progressed. He suggested that the cause of progression in such cases was primarily the detachment of small pieces of joint cartilage as the result of vibration.

In the same issue of this journal F. Linow described four cases of injury resulting from the use of pneumatic tools for stone crushing. One was in the shoulder and two in the elbow and one necrosis of the lunate bone.

A similar case of degeneration of the carpus produced by use of a compressed-air drill was reported in 1937 by A. R. Jones.[15] His patient had used a jackhammer, a powerful drill which is held in both hands and which if not properly held may "kick back." The patient received such a kick in 1927 and was off duty for four days. An X-ray ten years later shows such crushing of the semilunar bone that all typical features were lost. The os magnum showed an old fracture and contained a large cyst. There were also degenerative cavities at the lower end of the radius. The author was unable to state whether this condition was due entirely to the injury or also in part to the cumulative effects of using the drill, since there was no history of any other injury to the hand.

Rostock suggests[16] that the joint injury produced depends upon the way the particular joint reacts to vibration. In joints of the hand, he says, there is always produced a condition of osteo-chondritis dissecans, especially of the lunate, or a "pseudo-arthritis" of the navicular bone. At the elbow this osteo-chondritis ("subchondrial necrosis") also develops, and is caused by the bone surfaces being pressed together. Since there is in addition prolonged tension of the musculature, an abnormal strain is put on to the points of muscle attachment, and periosteal proliferation also develops (especially in brachialis internus), which may also affect the joints. Thick bone proliferation in the form of "olecranon spurs" is of this nature. Less often the shoulder-joints are affected, and where this occurs the condition is one of ordinary traumatic osteo-arthritis. These conditions, although they may be found in other classes of workers, are, according to him, ten times more common in miners using pneumatic drills than any other. True joint changes, however, occurred in only 0·08 per cent. of the miners in his series of 833 joint injuries alleged to have occurred in men who had worked with pneumatic tools for a long period. He mentions that between 1929 and 1933 there were only 336 cases who received compensation under the Act for occupational joint injuries of this sort in all Germany.

A further type of joint injury caused by these tools was reported by Moulonguet[17] in 1931, but not apparently since. He described the formation of foreign bodies in the right elbow-joints of two quarrymen who had complained of stiffness and pain. There was no previous rheumatic history. One of them, aged

forty, had used the tool for three years, whilst the other, aged forty-five, had worked in this way for ten years. Figs. 1 and 2 are tracings of the original X-ray in the latter case.

My own contribution is the discovery in a roadman, aged forty-eight, of a calcified subacromial bursa in conjunction with early degenerative changes around the shoulder-joint. He had used the drill for a period of only four years, but had noticed gradually increasing stiffness and pain in the shoulder which he attributed to his work (Fig. 2). At the time of seeing him the

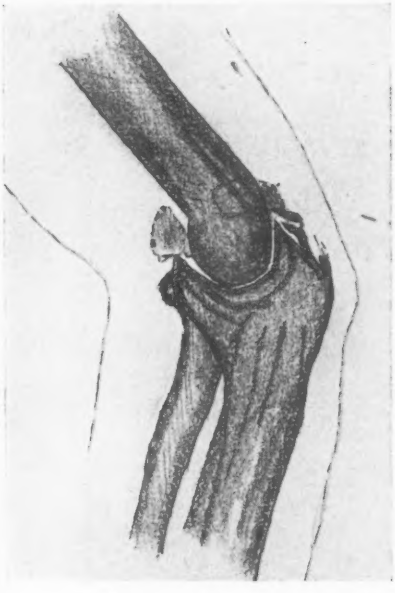

FIG. 1.

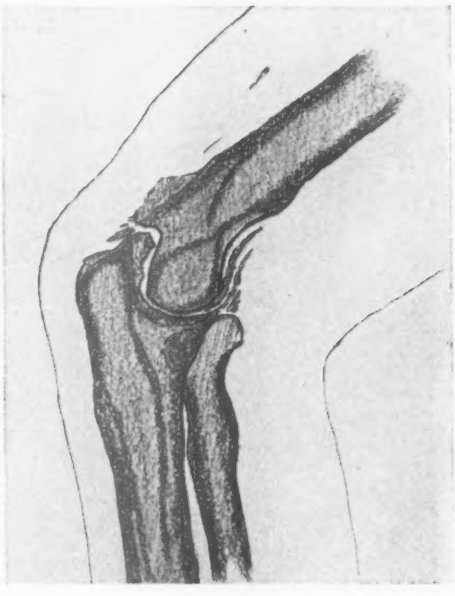

FIG. 2.

pain had become very acute, and he was unable to follow his employment. It seemed certain that this condition was intimately connected with the use of the pneumatic drill. I am unable, however, to find any record of another case of this type.

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STREPTOCOCCAL ANTIFIBRINOLYSIN IN RHEUMATOID ARTHRITIS AND SPONDYLITIS ANKYLOPOIETICA

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The presence of agglutinins to hæmolytic streptococci in the blood of many patients with rheumatoid arthritis was first reported by Cecil, Nicholls and Stainsby (1931). This phenomenon has been confirmed since by Dawson, Olmstead and Boots (1932), Nicholls and Stainsby (1933), Keefer, Myers and Oppel (1933), Cox and Hill (1934), Blair and Hallman (1935), McEwen, Chasis and Alexander (1935), and Neil and Hartung (1937); but the incidence of positive agglutination reactions varied from 85 per cent. (Blair and Hallman) to 30 per cent. (Neil and Hartung). Dawson, Olmstead and Boots (1932) suggested that the property of agglutinating hæmolytic streptococci is related to the age of the patient and the duration of the disease, and this was confirmed by Nicholls and Stainsby (1933). However, since all patients with rheumatoid arthritis do not show a positive agglutinin reaction, the exact significance of the finding is open to discussion. Attempts have been made to demonstrate the presence of other antibodies to the hæmolytic streptococcus in such patients. McEwen, Chasis and Alexander (1935) reported positive precipitin reactions between sera of patients with rheumatoid arthritis and various groups of hæmolytic streptococci, and Neil and Hartung (1937) found that the precipitin reaction roughly paralleled the agglutination, although the two did not necessarily occur together. Myers and Keefer (1934) confirmed the fact that the blood of patients recovering from hæmolytic streptococcal infections contains antistreptolysin in high titre, and that, while comparable titres are commonly found in patients with acute rheumatism, no such titre was found in patients with rheumatoid arthritis. On the other hand, antistreptolysin titres definitely above normal were found by Blair and Hallman (1935) in about one-third of the sera from similar patients. It has been shown that antifibrinolysin can

be demonstrated in the blood of some patients recovering from hæmolytic streptococcal infection (Tillett and Garner (1933), Tillett, Edwards and Garner (1934), Myers, Keefer and Holmes (1935), etc.). It is now generally agreed that antifibrinolysin similarly occurs in the blood of a considerable proportion of patients with acute rheumatism (Hadfield, Magee and Perry (1934), McEwen, Alexander and Bunim (1935), Myers, Keefer and Holmes (1935), etc.). In view of the occurrence of streptococcal agglutinins in the patients with rheumatoid arthritis already described, it was decided to investigate a series of patients with this disease for the presence of antifibrinolysin. Antifibrinolysin estimations were therefore made on the sera of thirty-three patients with typical rheumatoid arthritis. All the patients in this series had been ill for more than six months, and many for several years. Repeated observations were made during and after treatment with gold salts, and in all 171 observations were made on the thirty-three patients, only eight patients being tested once only. The technique was similar to that described by Tillett and Garner (1933), and every plasma was tested against four different strains of streptococci, as described by Perry (1939). Of the 171 observations, 161 fell within the normal range. In five tests on two of the patients normal at other times a doubtful degree of resistance of fibrinolysin was found (four times in one patient and once in the other). Definite resistance to fibrinolysin occurred only five times: twice in a patient normal at four other times and three times in a patient normal on eight other occasions.

Since spondylitis ankylopoietica presents many features in common with rheumatoid arthritis, blood from eight typical examples of this disease was also investigated, forty-nine observations being made on the eight patients. Of these, seven gave negative reactions whenever the blood was tested, and one gave a positive reaction when first examined, but was normal on two subsequent occasions.

These results are in striking contrast to those found in acute rheumatism, in the active stage of which antifibrinolysin was demonstrated in 75 per cent. of forty-four patients (Perry, 1939); and they show that streptococcal antifibrinolysin is not found at all commonly in the blood of patients suffering from rheumatoid arthritis or spondylitis ankylopoietica.

SUMMARY

Antifibrinolysin determinations were carried out on the plasma of thirty-three patients with rheumatoid arthritis. Definite resistance to fibrinolysin was found as a transient phenomenon in two of the patients. Similar tests were performed forty-nine times in eight patients with spondylitis ankylopoietica. A transient rise in the antifibrinolysin titre was found in one patient only.

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FOURTH ANNUAL REPORT OF THE EMPIRE RHEUMATISM COUNCIL

November, 1939, *to November*, 1940

My Lords, Ladies and Gentlemen,

This year, again, I shall not have the pleasure of addressing you personally, as it has been considered inadvisable to call members of the Council together in London for the Annual Meeting. I submit to you the Annual Report to October 31, 1940, repeating the assurance given last year that if any member wishes to bring up a matter arising out of the Report prompt attention will be given to it by the War Emergency Committee.

During twelve months of resistance to the Nazi attempt to plunge the world again into a Dark Age, the full brunt of which has fallen on these islands, the decision of 1939 that our Council should carry on its work has been justified. Difficulties and even dangers have been encountered, but with one exception the research tasks undertaken were continued until almost the close of the year.

Substantial progress has been made with plans for a national scheme of treatment, and this is now almost ready for publication. We may claim, too, the moral advantage of carrying on a campaign such as ours during this mortal struggle. This fact indicates the sober confidence of the British peoples that our humane civilisation is not doomed to perish and that they will come out of this ordeal strong enough, and wise enough, to give a lead in building up a better world. In that task medical research has a great part to play in bringing under control those secular enemies of mankind, the agencies of disease. It is fitting that its workers should keep to their tasks, following the high example of doctor and nurse who stand by their patients in the shattered hospitals. The healing institutions seem to be the favoured objectives of a malignant barbarism.

Work for the Defence Forces

Throughout the year our Council has taken every opportunity that offered of service to the Defence Forces.

Our Naval Research Foundation has continued its work,

with headquarters at the emergency station of the Royal Naval Medical School. Its Director, Dr. C. A. Green, has volunteered his assistance to the Navy in any pathological tasks arising out of the war, over and above his investigations into Rheumatic Disease.

We had a welcome invitation from the Royal Air Force Medical Service to help in their plans for treatment of Rheumatic Disease; and promptly responded.

In respect of the Army, the Empire Rheumatism Council has sought, for reasons of the well-being of the troops and of economising in national expenditure, the establishment of specialised rheumatism hospitals. A high incidence of Rheumatic Disease among the soldiers is probable; apart from other conditions of service, the intensive development of mechanised warfare will make them subject to conditions known to be specially productive of Rheumatic Disease in industrial life—frequent small bruising and tearing injuries, working in cramped conditions, frequent alternations of heat and cold. Since it is only recently that the medical profession has given specialised attention to the problems of rheumatism, and the number of medical practitioners with wide experience of its diagnosis and treatment is limited, it would be wise to concentrate their expert knowledge in specialised hospitals.

A plan has been submitted and the offer made of the fullest measure of help from our Council. There is an administrative problem presented here: that the physical treatments which are necessary in the treatment of Rheumatic Disease are also frequently called for in the rehabilitation of joints, muscles and nerves injured by wounds. It would be advisable, therefore, not to duplicate the medical and auxiliary personnel or the technical equipment of hospitals for the treatment of such conditions, whether arising from wounds or from Rheumatic Disease.

We may confidently trust that a solution of the problem will be found; that it will not be forgotten that a very large proportion of the cases to be treated will be rheumatic, and that rheumatism is primarily the care of the physician, though in many cases valuable help can be given by the orthopædic surgeon.

Whilst with the B.E.F. in France our hon. medical secretary, Lieut.-Colonel W. S. C. Copeman, was permitted to establish a special rheumatism ward at one of the Base Hospitals, and it was intended that a special Rheumatism Hospital would be set up in the course of time. The withdrawal of the Army from

France stopped this development. Lieut.-Colonel Copeman's paper ("Notes on Treatment of Rheumatic Diseases in the B.E.F.", *Journal of the R.A.M.C.*, May, 1940), with its notes on the contrivance of physical treatment apparatus from improvised material available in the field from Army stores, put into practical effect the contention of our Council that grappling with rheumatism on a national scale will not prove a vastly expensive undertaking if there are applied commonsense methods guided by scientific principles.

Lieut.-Colonel W. S. C. Copeman, who is now commanding the medical division of one of the military hospitals in the Southern Region, has been informed by the War Office that funds have been made available for the establishment of an Occupational Therapy unit in this hospital. It is to be for the treatment of sufferers from rheumatism and from the after-results of war injuries.

Our Council has offered help to the Australian Army medical authorities in dealing with the incidence of Rheumatic Disease in the Australian Forces. Lieut.-Colonel Anderson, A.D.G.M.S., expressed his appreciation of the offer and, at his request, a triplicate set of documents embodying the Council's views on war treatment of rheumatism has been sent to him for transmission to Australia, to the Australian Expeditionary Force, and to the Australian contingent in Great Britain.

Perhaps the heading of this section of our Report implies a distinction which really has ceased to exist in this war. "Defence Forces" now comprise the whole community, excluding only the totally infirm and the children. Otherwise every man and woman is in the front line of battle, subject to almost equal dangers and hardships as the serving men, facing them generally with an equal degree of heroism and "doing their bit" with cheerfulness and resourcefulness. Thus work to safeguard civilians from the scourge of rheumatism (and of other diseases) and from its aggravation by war conditions is in a true sense Defence work.

It is my privilege to have been invited by the Government to accept the chairmanship of the Medical Advisory Committee of the Ministry of Labour and National Service, and of the Committee of Inquiry regarding health conditions in air-raid shelters. Both these Committees hold key positions in relation to the War on Rheumatism, and it will be my duty to keep them in close touch with the Empire Rheumatism Council.

Research

As already noted, the work of the Naval Research Foundation (Dr. C. A. Green, Director) has continued at the temporary centre of the Royal Naval Medical School; and a section at the West London Hospital (Professor Ernst Freund, Director). Progress reports are encouraging.

The work of the Sir Alexander Maclean Laboratory at the St. John Clinic (Dr. H. J. Taylor, Director) was continued with good progress.

The work of the other Sir Alexander Maclean Laboratory at the Hospital of St. John and St. Elizabeth (Dr. C. B. Dyson, Director) is still suspended.

Sir Alexander Maclean, in a spirit of confidence which is deeply appreciated, has intimated his willingness that funds from his generous gift can be diverted, as and when necessary, from the original objectives to any work for the Defence Services undertaken by us.

The work of Dr. E. G. L. Bywaters (in conjunction with a Beit Fellowship) at the British Post-Graduate Medical School, University of London, was interrupted in September. Dr. Bywaters' research work on rheumatism was changed over to investigation of "Shock", for which there was an urgent call.

Dr. Cecilia Lutwak-Mann during the year continued her work at the University of Cambridge School of Biochemistry with encouraging results.

Dr. Francis J. Bach has carried out, on behalf of our Council, an investigation of a system of treatment advocated by Dr. G. Laughton Scott. A preliminary report has been presented and a final report is expected at an early date.

The initiative of the Empire Rheumatism Council in establishing (1939), with a grant from the Giff-Edmonds Trust, at the Hospital of St. John and St. Elizabeth, a department for clinical research into Occupational Therapy in the treatment of arthritis has proved of significant value. Though the work there was interrupted by war conditions it proved to be a successful pioneer effort to draw attention to the value of this means of rehabilitating disabled joints and muscles. Occupational Therapy promises to become established as a standard method in rheumatism treatment centres. Already, as noted before, the War Office have sanctioned it for the Army.

An interesting note in the field of clinical research has come from the Rheumatism Department of the West London Hospital (which in the absence of Lieut.-Colonel Copeman was carried on by Dr. Hugh Burt and, later, by Dr. O. Savage, assisted by Dr. Elkin). The latest report on its work states: "The results obtained at the West London Hospital suggest that, in a number of cases of arthritis, treatment as an out-patient is more beneficial than as an in-patient . . . The latter makes some patients too reliant on bed and therefore has a weakening effect."

Members of the Council will note that our Annual Reports do not reprint the lengthy progress records of the numerous research investigations which are being carried on by workers of our Council. The reason is economy in printing. The chief of these records are printed in our official journal, *Annals of the Rheumatic Diseases*, of which five numbers have been published and a sixth will be issued shortly. (Back numbers of this journal are available. Those who are interested can obtain them from the publishers, H. K. Lewis and Co., London, or from the office of the Empire Rheumatism Council.) Other records have been published in the Medical Press.

Treatment

The reopening of the British Red Cross Clinic for Rheumatism (Dr. H. Gordon Thompson, Administrator), Peto Place, in February, 1940, was a most welcome restoration of treatment facilities in the London area. The Clinic quickly had a full list of patients and evening sessions had to be arranged.

The treatment work at the Clinic of the Order of St. John (Sir Leonard Hill, Administrator) was carried on.

The West London Hospital Rheumatism Clinic reopened April, 1940, and has continued its work since.

Administration

The War Emergency Committee has held meetings whenever necessary, and in the intervals the officials of the Council have kept in constant touch with its members. The Committee has been strengthened by the co-option of Colonel the Rt. Hon. John Gretton, P.C., M.P., Dr. C. W. Buckley, F.R.C.P., and Colonel F. D. Howitt, C.V.O., F.R.C.P.

The office of the Council has been maintained. As it is in a somewhat dangerous area the more important financial documents of the Council have been deposited with our Bank, and the chief files moved to an auxiliary office at 87, Grosvenor Avenue, Carshalton. This has not involved any extra expense to the Council, the necessary accommodation having been given by the assistant secretary.

I know that the Council will wish me to place on record again the devoted zeal which members of your War Emergency Committee have shown throughout the year. Members of the Committee are: Sir William Willcox, K.C.I.E., F.R.C.P.; A. G. Timbrell Fisher, F.R.C.S., M.C.; Mervyn H. Gordon, C.M.G., F.R.S., D.M.; Sir Walter Kinnear, K.B.E.; T. W. Robinson, A.C.A.; Colonel the Rt. Hon. John Gretton, P.C., M.P.; Lieut.-Colonel W. S. C. Copeman, F.R.C.P.; C. W. Buckley, M.D., F.R.C.P.; Colonel F. D. Howitt, C.V.O., F.R.C.P.

Whilst reasonable prudence is being observed, "alert" signals have never interrupted work. It has been the experience, I think, of most members of the Committee to have been engaged on the Council's affairs during raid warnings. To the two officials of the Council, Sir Frank Fox and Miss V. C. Small, we owe our very best thanks. No task has been neglected by them, or seriously delayed, in spite of temporary difficulties of travel and of other means of communication.

I leave to the Finance members of the War Emergency Committee to report in some detail on our financial position and will only congratulate them on the fact that we have passed through a troubled year and may expect to pass through the coming year—if it should unhappily prove to be also a war year—without difficulty. Careful financial policy in the past made this possible.

Considering the circumstances, there has been a fair harvest of publicity during the year for the work of the Council, in the lay as well as in the medical press. Undoubtedly public interest in rheumatism has been awakened effectively during the past few years. With Western Europe, where humane civilisation is for the time being in a prison house, there have been no communications since the spring, but correspondence with the United States, the Dominions and Colonies (and, to a lesser extent, with South America) has been considerable. Indicative of the extent to which knowledge of the work of the Council has spread

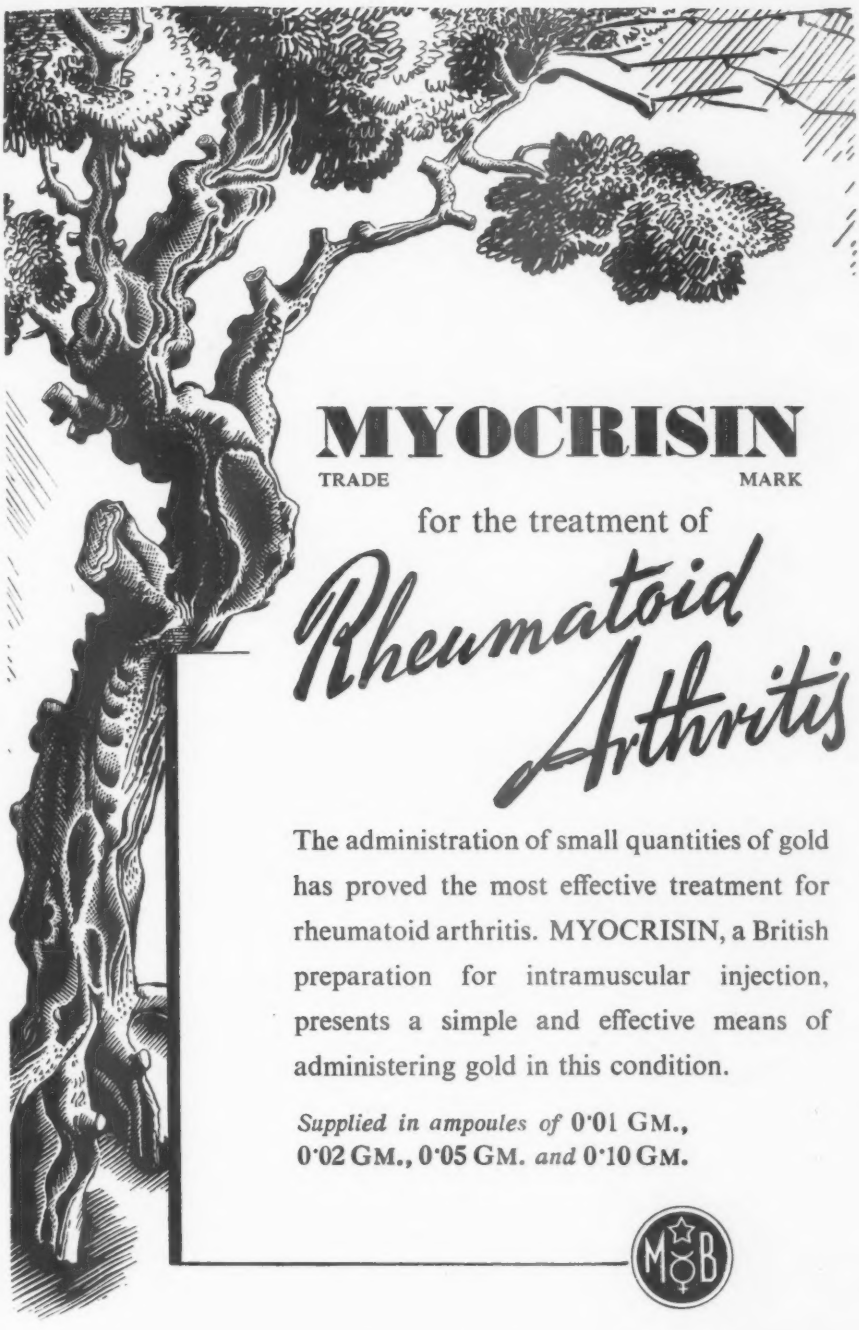
is the arrival from time to time of letters asking for advice from lonely corners of the earth, such as Central Mexico.

No general appeal has been attempted during the year, but every opportunity has been taken to bring our good cause to the attention of likely sympathisers, especially the Approved Societies; in this matter Sir Walter Kinnear has given great help.

The Future

We can, I feel, look back on the work of the past four years —work tragically hindered by war and preparations for war— with some satisfaction. Research into causative factors has progressed. Clinical research into methods of treatment has made notable advances. We are far better equipped than we were in 1936 to advise the community as to how the ravages of Rheumatic Disease can be checked at once, and have made distinct advance towards the objective of effective control. Such effective control, when it comes to be realised, will remove from humanity one of its greatest afflictions, bringing us nearer to understanding the vision of Isaiah: "They shall not hurt nor destroy in all my holy mountain: for the earth shall be full of the knowledge of the Lord, as the waters cover the sea."

(*Signed*) HORDER.



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